EAST Search History

Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
L1	918	(ethyl adj nitrite) or n2o or (nitrosyl adj ethoxide) or (nitrous adj ether) or (nitrous adj ethyl adj ether) or (sweet adj spirit adj niter)	US-PGPUB; USPAT	OR	OFF	2006/03/20 08:52
L2	153796	co2 or (carbon adj dioxide) or (carbonic adj acid adj gas) or (carbon-12c adj dioxide-1602) or (carbon-12 adj dioxide) or (r adj "744") or (khladon adj "744") or (carbonic adj acid adj anhydride)	US-PGPUB; USPAT	OR	OFF	2006/03/20 08:50
L3	257	l1 and l2	US-PGPUB; USPAT	OR	OFF	2006/03/20 08:50
L4	70	l3 and (blood or hemoglobin or hem? or hypoxic or hypoxemia)	US-PGPUB; USPAT	OR	OFF	2006/03/20 08:50
L5	43	l1 and (blood adj flow)	US-PGPUB; USPAT	OR	OFF	2006/03/20 08:53
L6	32	l5 and (abdominal or liver or kidney or hepatic or renal or intestine or intestinal or percardium or organ)	US-PGPUB; USPAT	OR	OFF	2006/03/20 08:53

PALM INTRANET

Day : Monday
Date: 3/20/2006
Time: 10:33:59

Inventor Information for 10/714980

Inventor Name	City	State/Country
STAMLER, JONATHAN S.	CHAPEL HILL	NORTH CAROLINA

Appln Info | Contents | Petition Info | Atty/Agent Info | Continuity Data | Foreign Data | **Inventors**

Search Another: Application# [ ] Search **or Patent#** [ ] Search

PCT / [ ] / [ ] Search **or PG PUBS #** [ ] Search

Attorney Docket # [ ] Search

Bar Code # [ ] Search

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Back to PALM | ASSIGNMENT | OASIS | Home page

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LOGINID:SSSPTA1616BSK

PASSWORD:
TERMINAL (ENTER 1, 2, 3, OR ?):2

* * * * * * * * * * Welcome to STN International * * * * * * * * * *

NEWS 1		Web Page URLs for STN Seminar Schedule - N. America
NEWS 2		"Ask CAS" for self-help around the clock
NEWS 3	DEC 21	IPC search and display fields enhanced in CA/CAplus with the IPC reform
NEWS 4	DEC 23	New IPC8 SEARCH, DISPLAY, and SELECT fields in USPATFULL/ USPAT2
NEWS 5	JAN 13	IPC 8 searching in IFIPAT, IFIUDB, and IFICDB
NEWS 6	JAN 13	New IPC 8 SEARCH, DISPLAY, and SELECT enhancements added to INPADOC
NEWS 7	JAN 17	Pre-1988 INPI data added to MARPAT
NEWS 8	JAN 17	IPC 8 in the WPI family of databases including WPIFV
NEWS 9	JAN 30	Saved answer limit increased
NEWS 10	JAN 31	Monthly current-awareness alert (SDI) frequency added to TULSA
NEWS 11	FEB 21	STN AnaVist, Version 1.1, lets you share your STN AnaVist visualization results
NEWS 12	FEB 22	Status of current WO (PCT) information on STN
NEWS 13	FEB 22	The IPC thesaurus added to additional patent databases on STN
NEWS 14	FEB 22	Updates in EPFULL; IPC 8 enhancements added
NEWS 15	FEB 27	New STN AnaVist pricing effective March 1, 2006
NEWS 16	FEB 28	MEDLINE/LMEDLINE reload improves functionality
NEWS 17	FEB 28	TOXCENTER reloaded with enhancements
NEWS 18	FEB 28	REGISTRY/ZREGISTRY enhanced with more experimental spectral property data
NEWS 19	MAR 01	INSPEC reloaded and enhanced
NEWS 20	MAR 03	Updates in PATDPA; addition of IPC 8 data without attributes
NEWS 21	MAR 08	X.25 communication option no longer available after June 2006

NEWS EXPRESS FEBRUARY 15 CURRENT VERSION FOR WINDOWS IS V8.01a,
CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
AND CURRENT DISCOVER FILE IS DATED 19 DECEMBER 2005.
V8.0 AND V8.01 USERS CAN OBTAIN THE UPGRADE TO V8.01a AT
http://download.cas.org/express/v8.0-Discover/

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* * * * * * * * * * * * * * STN Columbus * * * * * * * * * * * * * * * *

FILE 'HOME' ENTERED AT 08:16:30 ON 20 MAR 2006

=> file reg
COST IN U.S. DOLLARS SINCE FILE ENTRY TOTAL SESSION

FULL ESTIMATED COST 0.21 0.21

FILE 'REGISTRY' ENTERED AT 08:16:38 ON 20 MAR 2006
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.

Property values tagged with IC are from the ZIC/VINITI data file
provided by InfoChem.

STRUCTURE FILE UPDATES: 17 MAR 2006 HIGHEST RN 877201-63-3
DICTIONARY FILE UPDATES: 17 MAR 2006 HIGHEST RN 877201-63-3

New CAS Information Use Policies, enter HELP USAGETERMS for details.

TSCA INFORMATION NOW CURRENT THROUGH January 6, 2006

Please note that search-term pricing does apply when
conducting SmartSELECT searches.

```
**********************************************************************
*                                                                    *
* The CA roles and document type information have been removed from *
* the IDE default display format and the ED field has been added,   *
* effective March 20, 2005.  A new display format, IDERL, is now    *
* available and contains the CA role and document type information. *
*                                                                    *
**********************************************************************
```

Structure search iteration limits have been increased. See HELP SLIMITS
for details.

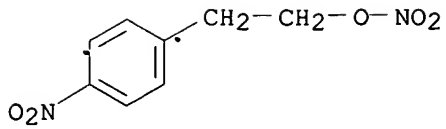
REGISTRY includes numerically searchable data for experimental and
predicted properties as well as tags indicating availability of
experimental property data in the original document. For information
on property searching in REGISTRY, refer to:

http://www.cas.org/ONLINE/UG/regprops.html

```
=> s ethyl nitrate
       7212148 ETHYL
            13 ETHYLS
       7212148 ETHYL
                 (ETHYL OR ETHYLS)
         44933 NITRATE
             7 NITRATES
         44933 NITRATE
                 (NITRATE OR NITRATES)
L1         474 ETHYL NITRATE
                 (ETHYL(W)NITRATE)

=> d 470-474

L1   ANSWER 470 OF 474  REGISTRY  COPYRIGHT 2006 ACS on STN
RN   1081-79-4  REGISTRY
ED   Entered STN:  16 Nov 1984
CN   Benzeneethanol, 4-nitro-, nitrate (ester) (9CI)  (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN   Phenethyl alcohol, p-nitro-, nitrate (6CI, 7CI)
CN   Phenethyl alcohol, p-nitro-, nitrate (ester) (8CI)
OTHER NAMES:
CN   2-(4-Nitrophenyl)ethyl nitrate
FS   3D CONCORD
MF   C8 H8 N2 O5
LC   STN Files:   BEILSTEIN*, CA, CAOLD, CAPLUS, CASREACT
        (*File contains numerically searchable property data)
```



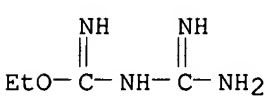
PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

18 REFERENCES IN FILE CA (1907 TO DATE)
18 REFERENCES IN FILE CAPLUS (1907 TO DATE)
6 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L1 ANSWER 471 OF 474 REGISTRY COPYRIGHT 2006 ACS on STN
RN 761-96-6 REGISTRY
ED Entered STN: 16 Nov 1984
CN Carbamimidic acid, (aminoiminomethyl)-, ethyl ester, mononitrate (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN **Pseudourea, 3-amidino-2-ethyl-, nitrate (7CI, 8CI)**
MF C4 H10 N4 O . H N O3
LC STN Files: CA, CAOLD, CAPLUS

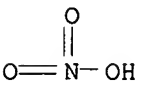
CM 1

CRN 41406-27-3
CMF C4 H10 N4 O



CM 2

CRN 7697-37-2
CMF H N O3



4 REFERENCES IN FILE CA (1907 TO DATE)
4 REFERENCES IN FILE CAPLUS (1907 TO DATE)
1 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L1 ANSWER 472 OF 474 REGISTRY COPYRIGHT 2006 ACS on STN
RN 646-02-6 REGISTRY
ED Entered STN: 16 Nov 1984
CN Ethanol, 2-amino-, nitrate (ester) (8CI, 9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Ethanol, 2-amino-, nitrate (7CI)
OTHER NAMES:
CN **2-Aminoethyl nitrate**
CN 2-Nitratoethylamine
CN **Aminoethyl nitrate**
CN Itramin
CN Monoethanolamine nitrate ester
CN Nitrolamine
FS 3D CONCORD
MF C2 H6 N2 O3
CI COM
LC STN Files: BEILSTEIN*, BIOSIS, CA, CAOLD, CAPLUS, CASREACT, DDFU, DRUGU, IFICDB, IFIPAT, IFIUDB, NIOSHTIC, TOXCENTER, USAN, USPAT2, USPATFULL

(*File contains numerically searchable property data)
Other Sources: WHO

PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

32 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
32 REFERENCES IN FILE CAPLUS (1907 TO DATE)
2 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L1 ANSWER 473 OF 474 REGISTRY COPYRIGHT 2006 ACS on STN
RN 625-58-1 REGISTRY
ED Entered STN: 16 Nov 1984
CN Nitric acid, ethyl ester (8CI, 9CI) (CA INDEX NAME)
OTHER NAMES:
CN **Ethyl nitrate**
CN NSC 8826
FS 3D CONCORD
MF C2 H5 N O3
CI COM
LC STN Files: ANABSTR, BEILSTEIN*, BIOSIS, CA, CAOLD, CAPLUS, CASREACT, CHEMINFORMRX, CHEMLIST, CHEMSAFE, CSNB, DETHERM*, GMELIN*, HSDB*, IFICDB, IFIPAT, IFIUDB, MEDLINE, MSDS-OHS, NIOSHTIC, PIRA, RTECS*, SPECINFO, TOXCENTER, USPATFULL
(*File contains numerically searchable property data)
Other Sources: EINECS**, NDSL**, TSCA**
(**Enter CHEMLIST File for up-to-date regulatory information)

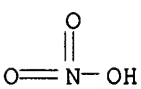
PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

550 REFERENCES IN FILE CA (1907 TO DATE)
2 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
551 REFERENCES IN FILE CAPLUS (1907 TO DATE)
7 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L1 ANSWER 474 OF 474 REGISTRY COPYRIGHT 2006 ACS on STN
RN 461-41-6 REGISTRY
ED Entered STN: 16 Nov 1984
CN **Ethanamine, 2,2,2-trifluoro-N-(2,2,2-trifluoroethyl)-, nitrate (9CI)** (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Diethylamine, 2,2,2,2',2',2'-hexafluoro-, nitrate (8CI)
MF C4 H5 F6 N . H N O3
LC STN Files: BEILSTEIN*, CA, CAOLD, CAPLUS
(*File contains numerically searchable property data)

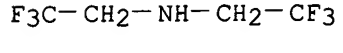
CM 1

CRN 7697-37-2
CMF H N O3



CM 2

CRN 407-01-2
CMF C4 H5 F6 N

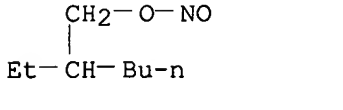


1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)
1 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

=> s ethyl nitrite
7212148 ETHYL
13 ETHYLS
7212148 ETHYL
(ETHYL OR ETHYLS)
3759 NITRITE
2 NITRITES
3759 NITRITE
(NITRITE OR NITRITES)
L2 52 ETHYL NITRITE
(ETHYL(W)NITRITE)

=> d 50-52

L2 ANSWER 50 OF 52 REGISTRY COPYRIGHT 2006 ACS on STN
RN 1653-42-5 REGISTRY
ED Entered STN: 16 Nov 1984
CN Nitrous acid, 2-ethylhexyl ester (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN **1-Hexanol, 2-ethyl-, nitrite (7CI, 8CI)**
OTHER NAMES:
CN 2-Ethylhexyl nitrite
FS 3D CONCORD
MF C8 H17 N O2
LC STN Files: BEILSTEIN*, CA, CAOLD, CAPLUS, CHEMLIST, TOXCENTER, USPATFULL
(*File contains numerically searchable property data)
Other Sources: EINECS**
(**Enter CHEMLIST File for up-to-date regulatory information)



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

12 REFERENCES IN FILE CA (1907 TO DATE)
12 REFERENCES IN FILE CAPLUS (1907 TO DATE)
3 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L2 ANSWER 51 OF 52 REGISTRY COPYRIGHT 2006 ACS on STN
RN 540-80-7 REGISTRY
ED Entered STN: 16 Nov 1984
CN Nitrous acid, 1,1-dimethylethyl ester (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Nitrous acid, tert-butyl ester (8CI)
CN tert-Butyl nitrite (6CI, 7CI)
OTHER NAMES:
CN **α,α-Dimethylethyl nitrite**
CN **1,1-Dimethylethyl nitrite**
FS 3D CONCORD
MF C4 H9 N O2
CI COM
LC STN Files: BEILSTEIN*, BIOSIS, CA, CAOLD, CAPLUS, CASREACT, CHEMCATS,

CHEMINFORMRX, CHEMLIST, CSCHEM, CSNB, DETHERM*, GMELIN*, HSDB*, IFICDB, IFIPAT, IFIUDB, MRCK*, MSDS-OHS, PS, RTECS*, TOXCENTER, USPAT2, USPATFULL
(*File contains numerically searchable property data)
Other Sources: EINECS**
(**Enter CHEMLIST File for up-to-date regulatory information)

t-Bu—O—NO

PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

378 REFERENCES IN FILE CA (1907 TO DATE)
5 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
378 REFERENCES IN FILE CAPLUS (1907 TO DATE)
29 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L2 ANSWER 52 OF 52 REGISTRY COPYRIGHT 2006 ACS on STN
RN 109-95-5 REGISTRY
ED Entered STN: 16 Nov 1984
CN Nitrous acid, ethyl ester (8CI, 9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN **Ethyl nitrite (6CI, 7CI)**
OTHER NAMES:
CN N2O
CN Nitrosyl ethoxide
CN Nitrous ether
CN Nitrous ethyl ether
CN **Spirit of ethyl nitrite**
CN Sweet spirit of niter
FS 3D CONCORD
DR 8013-58-9
MF C2 H5 N O2
CI COM
LC STN Files: ADISNEWS, AGRICOLA, BEILSTEIN*, BIOSIS, CA, CAOLD, CAPLUS, CASREACT, CHEMCATS, CHEMINFORMRX, CHEMLIST, CHEMSAFE, CIN, CSCHEM, CSNB, DETHERM*, DIOGENES, EMBASE, GMELIN*, HSDB*, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MRCK*, MSDS-OHS, NIOSHTIC, PDLCOM*, PIRA, PROMT, RTECS*, TOXCENTER, USAN, USPAT2, USPATFULL
(*File contains numerically searchable property data)
Other Sources: DSL**, EINECS**, TSCA**
(**Enter CHEMLIST File for up-to-date regulatory information)

$H_3C—CH_2—O—N{=}O$

PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

557 REFERENCES IN FILE CA (1907 TO DATE)
2 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
557 REFERENCES IN FILE CAPLUS (1907 TO DATE)
39 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

=> file caplus medline biosis embase

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	35.12	35.33

FILE 'CAPLUS' ENTERED AT 08:17:46 ON 20 MAR 2006
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FILE 'MEDLINE' ENTERED AT 08:17:46 ON 20 MAR 2006

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FILE 'EMBASE' ENTERED AT 08:17:46 ON 20 MAR 2006
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=> s ethyl nitrite or n2o or nitrosyl ethoxide or nitrous ether or nitrous ethyl ether or
spirit of ethyl nitrite or sweet spirit of niter
L3       45910 ETHYL NITRITE OR N2O OR NITROSYL ETHOXIDE OR NITROUS ETHER OR
               NITROUS ETHYL ETHER OR SPIRIT OF ETHYL NITRITE OR SWEET SPIRIT
              OF NITER

=>

=> s 109-95-5/rn or 8013-58-5/rn
'RN' IS NOT A VALID FIELD CODE
'RN' IS NOT A VALID FIELD CODE
'RN' IS NOT A VALID FIELD CODE
L4         555 109-95-5/RN OR 8013-58-5/RN

=> s l4 or l3
L5       46026 L4 OR L3

=> file reg
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                         ENTRY    SESSION
FULL ESTIMATED COST                                      42.15      77.48

FILE 'REGISTRY' ENTERED AT 08:19:16 ON 20 MAR 2006
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STRUCTURE FILE UPDATES:   17 MAR 2006  HIGHEST RN 877201-63-3
DICTIONARY FILE UPDATES:  17 MAR 2006  HIGHEST RN 877201-63-3

New CAS Information Use Policies, enter HELP USAGETERMS for details.

TSCA INFORMATION NOW CURRENT THROUGH January 6, 2006

  Please note that search-term pricing does apply when
  conducting SmartSELECT searches.

**********************************************************************
*                                                                    *
* The CA roles and document type information have been removed from *
* the IDE default display format and the ED field has been added,   *
* effective March 20, 2005.  A new display format, IDERL, is now    *
* available and contains the CA role and document type information. *
*                                                                    *
**********************************************************************

Structure search iteration limits have been increased.  See HELP SLIMITS
for details.

REGISTRY includes numerically searchable data for experimental and
predicted properties as well as tags indicating availability of
experimental property data in the original document.  For information
on property searching in REGISTRY, refer to:

http://www.cas.org/ONLINE/UG/regprops.html

=> s carbon dioxide
        108821 CARBON
           305 CARBONS
```

```
        108821 CARBON
                 (CARBON OR CARBONS)
        116737 DIOXIDE
            17 DIOXIDES
        116737 DIOXIDE
                 (DIOXIDE OR DIOXIDES)
L6        1734 CARBON DIOXIDE
                 (CARBON(W)DIOXIDE)

=> d 1730-1734

L6   ANSWER 1730 OF 1734  REGISTRY  COPYRIGHT 2006 ACS on STN
RN   2537-69-1  REGISTRY
ED   Entered STN:  16 Nov 1984
CN   Carbon dioxide-18O2 (7CI, 8CI, 9CI)  (CA INDEX NAME)
OTHER NAMES:
CN   Carbon dioxide (12C18O2)
CN   Carbon dioxide (C18O2)
MF   C O2
CI   COM
LC   STN Files:   CA, CAOLD, CAPLUS, CASREACT, CHEMCATS, CSCHEM, GMELIN*,
       TOXCENTER, USPAT2, USPATFULL
         (*File contains numerically searchable property data)


18O=C=18O


**PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT**

             156 REFERENCES IN FILE CA (1907 TO DATE)
             156 REFERENCES IN FILE CAPLUS (1907 TO DATE)
              12 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L6   ANSWER 1731 OF 1734  REGISTRY  COPYRIGHT 2006 ACS on STN
RN   1554-45-6  REGISTRY
ED   Entered STN:  16 Nov 1984
CN   Hydrofluoric acid, compd. with carbon dioxide (1:1) (9CI)  (CA
     INDEX NAME)
OTHER CA INDEX NAMES:
CN   Carbon dioxide, compd. with hydrofluoric acid (1:1) (8CI, 9CI)
OTHER NAMES:
CN   Carbon dioxide compd. with hydrogen fluoride (1:1)
MF   C O2 . F H
LC   STN Files:   CA, CAOLD, CAPLUS, GMELIN*
         (*File contains numerically searchable property data)
CRN  (124-38-9)


O=C=O

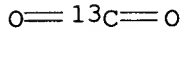

  ● HF


**PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT**

              11 REFERENCES IN FILE CA (1907 TO DATE)
              11 REFERENCES IN FILE CAPLUS (1907 TO DATE)
               1 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L6   ANSWER 1732 OF 1734  REGISTRY  COPYRIGHT 2006 ACS on STN
RN   1111-72-4  REGISTRY
ED   Entered STN:  16 Nov 1984
CN   Carbon-13C dioxide (6CI, 7CI, 8CI, 9CI)  (CA INDEX NAME)
OTHER NAMES:
```

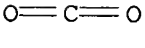
CN **Carbon dioxide (13C16O2)**
CN **Carbon dioxide (13CO2)**
CN **Carbon dioxide-13C**
CN **Carbon dioxide-C13**
CN Carbon-13 dioxide
CN **[13C]-Carbon dioxide**
MF C O2
CI COM
LC STN Files: AGRICOLA, ANABSTR, BIOSIS, CA, CAOLD, CAPLUS, CASREACT, CHEMCATS, CSCHEM, DETHERM*, GMELIN*, IFICDB, IFIPAT, IFIUDB, PROMT, TOXCENTER, USPAT2, USPATFULL
(*File contains numerically searchable property data)



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

886 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
889 REFERENCES IN FILE CAPLUS (1907 TO DATE)
17 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L6 ANSWER 1733 OF 1734 REGISTRY COPYRIGHT 2006 ACS on STN
RN 124-38-9 REGISTRY
ED Entered STN: 16 Nov 1984
CN **Carbon dioxide (8CI, 9CI)** (CA INDEX NAME)
OTHER NAMES:
CN Carbon oxide (CO2)
CN Carbon-12 dioxide
CN Carbon-12C dioxide-16O2
CN Carbonic acid anhydride
CN Carbonic acid gas
CN Carbonic anhydride
CN Dry ice
CN Khladon 744
CN R 744
FS 3D CONCORD
DR 18923-20-1
MF C O2
CI COM
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, BIOSIS, BIOTECHNO, CA, CABA, CAOLD, CAPLUS, CASREACT, CBNB, CHEMCATS, CHEMINFORMRX, CHEMLIST, CHEMSAFE, CIN, CSCHEM, CSNB, DDFU, DETHERM*, DIOGENES, DIPPR*, DRUGU, EMBASE, ENCOMPLIT, ENCOMPLIT2, ENCOMPPAT, ENCOMPPAT2, GMELIN*, HSDB*, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MRCK*, MSDS-OHS, NIOSHTIC, PDLCOM*, PIRA, PROMT, PS, RTECS*, SPECINFO, TOXCENTER, TULSA, ULIDAT, USAN, USPAT2, USPATFULL, VETU, VTB
(*File contains numerically searchable property data)
Other Sources: DSL**, EINECS**, TSCA**
(**Enter CHEMLIST File for up-to-date regulatory information)



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

197437 REFERENCES IN FILE CA (1907 TO DATE)
806 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
197728 REFERENCES IN FILE CAPLUS (1907 TO DATE)
21 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L6 ANSWER 1734 OF 1734 REGISTRY COPYRIGHT 2006 ACS on STN
RN 51-90-1 REGISTRY
ED Entered STN: 16 Nov 1984

CN Carbon-14C dioxide (8CI, 9CI) (CA INDEX NAME)
OTHER NAMES:
CN **Carbon dioxide (14CO2)**
CN **Carbon dioxide labeled with carbon-14**
CN **Carbon dioxide, isotope of mass 14**
CN **Carbon dioxide-14**
CN **Carbon dioxide-14C**
CN Carbon-14 dioxide
CN **[14C]Carbon dioxide**
MF C O2
CI COM
LC STN Files: AGRICOLA, ANABSTR, BIOSIS, BIOTECHNO, CA, CAPLUS, CASREACT, CHEMCATS, CSCHEM, EMBASE, GMELIN*, IFICDB, IFIPAT, IFIUDB, NIOSHTIC, TOXCENTER, USPAT2, USPATFULL
(*File contains numerically searchable property data)

903 REFERENCES IN FILE CA (1907 TO DATE)
905 REFERENCES IN FILE CAPLUS (1907 TO DATE)

=> s ethyl nitrite or n2o or nitrosyl ethoxide or nitrous ether or nitrous ethyl ether or spirit of ethyl nitrite or sweet spirit of niter
```
       7212148 ETHYL
            13 ETHYLS
       7212148 ETHYL
                 (ETHYL OR ETHYLS)
          3759 NITRITE
             2 NITRITES
          3759 NITRITE
                 (NITRITE OR NITRITES)
            52 ETHYL NITRITE
                 (ETHYL(W)NITRITE)
           340 N2O
         28021 NITROSYL
             1 NITROSYLS
         28021 NITROSYL
                 (NITROSYL OR NITROSYLS)
           415 ETHOXIDE
             2 ETHOXIDES
           415 ETHOXIDE
                 (ETHOXIDE OR ETHOXIDES)
             1 NITROSYL ETHOXIDE
                 (NITROSYL(W)ETHOXIDE)
          2227 NITROUS
         85315 ETHER
           983 ETHERS
         85315 ETHER
                 (ETHER OR ETHERS)
             1 NITROUS ETHER
                 (NITROUS(W)ETHER)
          2227 NITROUS
       7212148 ETHYL
            13 ETHYLS
       7212148 ETHYL
                 (ETHYL OR ETHYLS)
         85315 ETHER
           983 ETHERS
         85315 ETHER
                 (ETHER OR ETHERS)
             1 NITROUS ETHYL ETHER
                 (NITROUS(W)ETHYL(W)ETHER)
            94 SPIRIT
            11 SPIRITS
           100 SPIRIT
                 (SPIRIT OR SPIRITS)
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        127148 OF
            11 OFS
        127159 OF
                  (OF OR OFS)
       7212148 ETHYL
            13 ETHYLS
       7212148 ETHYL
                  (ETHYL OR ETHYLS)
          3759 NITRITE
             2 NITRITES
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                  (NITRITE OR NITRITES)
             1 SPIRIT OF ETHYL NITRITE
                  (SPIRIT(W)OF(W)ETHYL(W)NITRITE)
           739 SWEET
            94 SPIRIT
            11 SPIRITS
           100 SPIRIT
                  (SPIRIT OR SPIRITS)
        127148 OF
            11 OFS
        127159 OF
                  (OF OR OFS)
             5 NITER
             1 SWEET SPIRIT OF NITER
                  (SWEET(W)SPIRIT(W)OF(W)NITER)
L7           391 ETHYL NITRITE OR N2O OR NITROSYL ETHOXIDE OR NITROUS ETHER OR
                 NITROUS ETHYL ETHER OR SPIRIT OF ETHYL NITRITE OR SWEET SPIRIT
                OF NITER

=> file caplus medline biosis embase
COST IN U.S. DOLLARS                            SINCE FILE      TOTAL
                                                     ENTRY    SESSION
FULL ESTIMATED COST                                105.58     183.06

FILE 'CAPLUS' ENTERED AT 08:20:13 ON 20 MAR 2006
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=> s carbon dioxide or co2 or carbon oxide or carbon-12 dioxide or darbonic acid anhydride or
carbonic acid gas or carbonic anhydride or 124-38-9/rn
'RN' IS NOT A VALID FIELD CODE
'RN' IS NOT A VALID FIELD CODE
'RN' IS NOT A VALID FIELD CODE
L8       738508 CARBON DIOXIDE OR CO2 OR CARBON OXIDE OR CARBON-12 DIOXIDE OR
                 DARBONIC ACID ANHYDRIDE OR CARBONIC ACID GAS OR CARBONIC ANHYDRI
                DE OR 124-38-9/RN

=> d his

     (FILE 'HOME' ENTERED AT 08:16:30 ON 20 MAR 2006)

     FILE 'REGISTRY' ENTERED AT 08:16:38 ON 20 MAR 2006
L1            474 S ETHYL NITRATE
L2             52 S ETHYL NITRITE

     FILE 'CAPLUS, MEDLINE, BIOSIS, EMBASE' ENTERED AT 08:17:46 ON 20 MAR 2006
L3          45910 S ETHYL NITRITE OR N2O OR NITROSYL ETHOXIDE OR NITROUS ETHER OR
L4            555 S 109-95-5/RN OR 8013-58-5/RN
L5          46026 S L4 OR L3
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     FILE 'REGISTRY' ENTERED AT 08:19:16 ON 20 MAR 2006
L6   .     1734 S CARBON DIOXIDE
L7          391 S ETHYL NITRITE OR N2O OR NITROSYL ETHOXIDE OR NITROUS ETHER OR

     FILE 'CAPLUS, MEDLINE, BIOSIS, EMBASE' ENTERED AT 08:20:13 ON 20 MAR 2006
L8       738508 S CARBON DIOXIDE OR CO2 OR CARBON OXIDE OR CARBON-12 DIOXIDE OR

=> s l8 and l5
L9         9502 L8 AND L5

=> s l9 and (blood or hemo? or hem? or hemoglobin or oxygen)_
MISSING OPERATOR OXYGEN)_
The search profile that was entered contains terms or
nested terms that are not separated by a logical operator.

=> s l9 and (blood or hem? or hemo? or oxygen)
L10        3065 L9 AND (BLOOD OR HEM? OR HEMO? OR OXYGEN)

=> s l10 and (blood flow)
L11         290 L10 AND (BLOOD FLOW)

=> dup rem l11
PROCESSING COMPLETED FOR L11
L12         181 DUP REM L11 (109 DUPLICATES REMOVED)

=> focus
PROCESSING COMPLETED FOR L12
L13         181 FOCUS L12 1-

=> d ibib abs 1-30

L13  ANSWER 1 OF 181  CAPLUS  COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER:         1946:8799  CAPLUS
DOCUMENT NUMBER:          40:8799
ORIGINAL REFERENCE NO.:   40:1592a-c
TITLE:                    The effects of alterations in the arterial tensions of
                          carbon dioxide and oxygen
                          on the cerebral blood flow and
                          cerebral oxygen consumption of normal young
                          men
AUTHOR(S):                Kety, S. S.; Schmidt, C. F.
SOURCE:                   American Journal of Physiology (1945), 143, 33
                          CODEN: AJPHAP; ISSN: 0002-9513
DOCUMENT TYPE:            Journal
LANGUAGE:                 Unavailable
AB   By means of the N2O method quant. measurements were made of
     blood flow and O consumption in the brains of
     unanesthetized healthy young men.  The cerebral blood
     flow and cerebral O consumption underwent the following changes,
     resp.: on active hyperventillation, -32% and 15%; on passive
     hyperventillation, -36% and 0%; on inhalation of 5% CO2, 58% and
     9%; on inhalation of 7% CO2, 125% and -8%; on inhalation of 85
     to 100% O, -12% and 0%; and on inhalation of 10% O, 48% and -2%.  For 5
     subjects the mean cerebral blood flow was estimated to
     average 927 cc./min., 20% of the cardiac output; and the mean cerebral O
     consumption to average 62 cc./min., 24% of the total O intake by the body.

L13  ANSWER 2 OF 181  CAPLUS  COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER:         1946:8798  CAPLUS
DOCUMENT NUMBER:          40:8798
ORIGINAL REFERENCE NO.:   40:1592a-c
TITLE:                    The effects of alterations in the arterial tensions of
                          carbon dioxide and oxygen
                          on the cerebral blood flow and
                          cerebral oxygen consumption of normal young
                          men
AUTHOR(S):                Kety, S. S.; Schmidt, C. F.
CORPORATE SOURCE:         Univ. of Penn., Philadelphia
```

SOURCE: American Journal of the Medical Sciences (1945), 210, 809-10
CODEN: AJMSA9; ISSN: 0002-9629
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable
AB By means of the **N2O** method quant. measurements were made of **blood flow** and O consumption in the brains of unanesthetized healthy young men. The cerebral **blood flow** and cerebral O consumption underwent the following changes, resp.: on active hyperventillation, -32% and 15%; on passive hyperventillation, -36% and 0%; on inhalation of 5% **CO2**, 58% and 9%; on inhalation of 7% **CO2**, 125% and -8%; on inhalation of 85 to 100% O, -12% and 0%; and on inhalation of 10% O, 48% and -2%. For 5 subjects the mean cerebral **blood flow** was estimated to average 927 cc./min., 20% of the cardiac output; and the mean cerebral O consumption to average 62 cc./min., 24% of the total O intake by the body.

L13 ANSWER 3 OF 181 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:117679 CAPLUS
DOCUMENT NUMBER: 138:163544
TITLE: Use of a **blood-flow** decrease preventing agent in conjunction with insufflating gas
INVENTOR(S): Stamler, Jonathan S.
PATENT ASSIGNEE(S): Duke University, USA
SOURCE: PCT Int. Appl., 16 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003011384	A1	20030213	WO 2002-US19810	20020712
W: AU, CA, JP				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR				
US 2003032917	A1	20030213	US 2001-919931	20010802
US 6676855	B2	20040113		
CA 2452529	AA	20030213	CA 2002-2452529	20020712
EP 1420851	A1	20040526	EP 2002-746630	20020712
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY, TR, BG, CZ, EE, SK				
JP 2004536878	T2	20041209	JP 2003-516614	20020712

PRIORITY APPLN. INFO.: US 2001-919931 A 20010802
WO 2002-US19810 W 20020712

AB A **blood-flow** decrease preventing agent is used to negate or reduce the decreased **oxygen** delivery in abdominal organs caused by insufflating gas. Preferably a gas is delivered into the abdominal cavity consisting essentially of the insufflating gas and the **blood-flow** decrease preventing agent. Very preferably, a gas is used consisting essentially of **carbon dioxide** as the insufflating gas and **Et nitrite** as the **blood-flow** to abdominal organ decrease preventing agent.
REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 4 OF 181 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:710451 CAPLUS
DOCUMENT NUMBER: 140:139244
TITLE: Effects of sevoflurane, propofol, and adjunct nitrous oxide on regional cerebral **blood flow**, **oxygen** consumption, and **blood** volume in humans
AUTHOR(S): Kaisti, Kaike K.; Langsjoe, Jaakko W.; Aalto, Sargo; Oikonen, Vesa; Sipilae, Hannu; Teraes, Mika; Hinkka, Susanna; Metsaehonkala, Liisa; Scheinin, Harry
CORPORATE SOURCE: Department of Anesthesiology, Turku University Hospital, Turku, Finland

SOURCE: Anesthesiology (2003), 99(3), 603-613
CODEN: ANESAV; ISSN: 0003-3022
PUBLISHER: Lippincott Williams & Wilkins
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Anesthetic agents, especially volatile anesthetics and nitrous oxide (**N2O**), are suspected to perturb cerebral homeostasis and vascular reactivity. The authors quantified the effects of sevoflurane and propofol as sole anesthetics and in combination with **N2O** on regional cerebral **blood flow** (rCBF), metabolic rate of **oxygen** (rCMRO2), and **blood** volume (rCBV) in the living human brain using positron emission tomog. 15O-labeled water, **oxygen**, and carbon monoxide were used as positron emission tomog. tracers to determine rCBF, rCMRO2 and rCBV, resp., in eight healthy male subjects during the awake state (baseline) and at four different anesthetic regimens: (1) sevoflurane alone, (2) sevoflurane plus 70% **N2O** (S+N), (3) propofol alone, and (4) propofol plus 70% **N2O** (P+N). Sevoflurane and propofol were titrated to keep a constant hypnotic depth (Bispectral Index 40) throughout anesthesia. End-tidal **carbon dioxide** was strictly kept at preinduction level. RESULTS: The mean ± SD end-tidal concentration of sevoflurane was 1.5 ± 0.3% during sevoflurane alone and 1.2 ± 0.3% during S+N ($P < 0.001$). The measured propofol concentration was 3.7 ± 0.7 μg/mL during propofol alone and 3.5 ± 0.7 μg/mL during P+N (not significant). Sevoflurane alone decreased rCBF in some (to 73-80% of baseline, $P < 0.01$), and propofol in all brain structures (to 53-70%, $P < 0.001$). Only propofol reduced also rCBV (in the cortex and cerebellum to 83-86% of baseline, $P < 0.05$). Both sevoflurane and propofol similarly reduced rCMRO2 in all brain areas to 56-70% and 50-68% of baseline, resp. ($P < 0.05$). The adjunct **N2O** counteracted some of the rCMRO2 and rCBF redns. caused by drugs alone, and especially during S+N, a widespread reduction ($P < 0.05$ for all cortex and cerebellum vs. awake) in the **oxygen** extraction fraction was seen. Adding of **N2O** did not alter the rCBV effects of sevoflurane and propofol alone. Propofol reduced rCBF and rCMRO2 comparably. Sevoflurane reduced rCBF less than propofol but rCMRO2 to an extent similar to propofol. These redns. in flow and metabolism were partly attenuated by adjunct **N2O**. S+N especially reduced the **oxygen** extraction fraction, suggesting disturbed flow-activity coupling in humans at a moderate depth of anesthesia.

REFERENCE COUNT: 58 THERE ARE 58 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 5 OF 181 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1992:584781 CAPLUS
DOCUMENT NUMBER: 117:184781
TITLE: The responsiveness of cerebral **blood flow** to changes in arterial **carbon dioxide** is maintained during propofol-nitrous oxide anesthesia in humans
AUTHOR(S): Fox, J.; Gelb, A. W.; Enns, J.; Murkin, J. M.; Farrar, J. K.; Manninen, P. H.
CORPORATE SOURCE: Dep. Anaesth., Univ. Hosp., London, ON, N6A 5A5, Can.
SOURCE: Anesthesiology (1992), 77(3), 453-6
CODEN: ANESAV; ISSN: 0003-3022
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Because it is common to manipulate PaCO2 during neurosurgery, it is essential to characterize the relationship between cerebral **blood flow** (CBF) and changes in PaCO2. The purpose of this study was to investigate the effects of propofol-**N2O** anesthesia on the CBF response to changes in PaCO2 in healthy subjects. In seven patients, anesthesia was induced with propofol 2.0-2.5 mg/kg and then maintained with a propofol infusion of 12 mg·kg-1·h-1 for 20 min mg·kg-1·h-1 for the remainder of the study. The subjects' lungs were ventilated with **N2O** in O2 (FIO2 0.3) to the end-tidal **CO2** present 133Xe and ten scintillation counters, five over each cerebral **hemisphere**. ETCO2 then was increased to 50 mmHg and CBF measurement repeated; ETCO2 then was reduced to 30 mmHg and CBF measurement repeated. Concurrent with each CBF measurement, arterial

blood was sampled for PaCO2 and **Hb** measurement. CBF at normocapnia (PaCO2 42 ± 2 mmHg) was 33 ± 7 mL·100 g-1·min-1, which increased to 58 ± 10 mL·100 g-1·min-1 and decreased to 19 ± 4 mL·100 g-1·min-1 on increasing PaCO2 (53 ± 4 mmHg) and CBF values were statistically different from those measured at any other time (CBF P < 0.002, PaCO2 P < 0.001). The slope of CBF vs. PaCO2 was 1.56 mL·100 g-1·min-1·mmHg. **Blood** pressure, temperature, and **Hb** were unchanged throughout the study. It is concluded that the cerebral vasculature in healthy subjects remains responsive to changes in PaCO2 during propofol-**N2O** anesthesia and that the slope of the CBF-**CO2** response is similar to that found in awake individuals and during anesthesia with other i.v. anesthetic agents.

L13 ANSWER 6 OF 181 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1998:566395 CAPLUS
DOCUMENT NUMBER: 129:310779
TITLE: Cerebral **blood flow** and **CO2** reactivity is similar during remifentanil/**N2O** and fentanyl/**N2O** anesthesia
AUTHOR(S): Ostapkovich, Noeleen D.; Baker, Kristy Z.; Fogarty-Mack, Patricia; Sisti, Michael B.; Young, William L.
CORPORATE SOURCE: College of Physicians and Surgeons, Columbia University, New York, NY, 10032, USA
SOURCE: Anesthesiology (1998), 89(2), 358-363
CODEN: ANESAV; ISSN: 0003-3022
PUBLISHER: Lippincott-Raven Publishers
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Remifentanil, a rapidly metabolized μ-opioid agonist, may offer advantages for neurosurgical procedures in which prolonged anesthetic effects can delay assessment of the patient. This study compared the effects of remifentanil-nitrous oxide on cerebral **blood flow** (CBF) and **carbon dioxide** reactivity with those of fentanyl-nitrous oxide anesthesia during craniotomy. After institutional approval and informed patient consent were obtained, 23 patients scheduled to undergo supratentorial tumor surgery were randomly assigned to remifentanil or fentanyl infusion groups in a double-blinded manner. Midazolam, thiopental, and pancuronium induction was followed by equipotent narcotic loading infusions of remifentanil (1 μg·kg-1·min-1) or fentanyl (2 μg·kg-1·min-1) for 5-10 min. Patients were ventilated with 2:1 nitrous oxide-**oxygen**, and opioid rates were reduced and then titrated to a stable **hemodynamic** effect. After dural exposure, CBF was measured by the i.v. 133xenon technique at normocapnia and hypocapnia. Reactivity of CBF to **carbon dioxide** was calculated as the absolute increase in CBF per mm of mercury increase in the partial pressure of **carbon dioxide** (PaCO2). Data were analyzed by repeated-measures anal. of variance, unpaired Student's t tests, or contingency anal. In the remifentanil group (n = 10), CBF decreased from 36±11 to 27±8 mL·100 g-1·min-1 as PaCO2 decreased from 33±5 to 25±2 mmHg. In the fentanyl group (n = 8), CBF decreased from 37±11 to 25±6 mL·100 g-1·min-1 as PaCO2 decreased from 34±3 to 25±3 mmHg. Absolute **carbon dioxide** reactivity was preserved with both agents: 1±1.2 mL · 100 g-1·min-1·mmHg-1 for remifentanil and 1.5±0.5 mL·100 g-1·min-1·mmHg-1 for fentanyl (P = 0.318). Remifentanil and fentanyl have similar effects on absolute CBF, and cerebrovascular **carbon dioxide** reactivity is maintained.
REFERENCE COUNT: 17 THERE ARE 17 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 7 OF 181 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1970:422007 CAPLUS
DOCUMENT NUMBER: 73:22007
TITLE: Gas-chromatographic analysis of **blood** gas in cerebral **blood flow** measurement by

the Kety and Schmidt method. Equilibration method with short elution time
AUTHOR(S): Becker, Klaus
CORPORATE SOURCE: Neurol. Klin., Justus Liebig-Univ., Giessen, Fed. Rep. Ger.
SOURCE: Klinische Wochenschrift (1970), 48(8), 464-7
CODEN: KLWOAZ; ISSN: 0023-2173
DOCUMENT TYPE: Journal
LANGUAGE: German
AB A modification of the method of Kety and Schmidt (1948) for the determination of cerebral **blood flow** in man is presented, in which simultaneous evaluation of O, N, **CO2**, and **N2O** is possible in a single gas-chromatographic run. Introduction of a special carrier loop compensates for tailing of the peaks due to the use of a reaction or equilibration chamber, and consequent reduced time of discharge makes it possible to get a good separation of the peaks by using a 2-column system. Standard curves for O and **CO2** are prepared with titrated solns. of H2O2 and NaHCO3. The reproducibility of the calibration curves is within 1%. This procedure permits a considerable acceleration and automation of **blood** gas anal. with a high degree of accuracy.

L13 ANSWER 8 OF 181 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:759944 CAPLUS
DOCUMENT NUMBER: 139:301927
TITLE: The effect of nitrous oxide on cerebrovascular reactivity to **carbon dioxide** in children during propofol anesthesia
AUTHOR(S): Karsli, C.; Wilson-Smith, E.; Luginbuehl, I.; Bissonnette, B.
CORPORATE SOURCE: Department of Anesthesia, The Hospital for Sick Children, ON, Can.
SOURCE: Anesthesia & Analgesia (Hagerstown, MD, United States) (2003), 97(3), 694-698
CODEN: AACRAT; ISSN: 0003-2999
PUBLISHER: Lippincott Williams & Wilkins
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Nitrous oxide (**N2O**) increases cerebral **blood flow** when used alone and in combination with propofol. We investigated the effects of **N2O** on cerebrovascular **CO2** reactivity (CCO2R) during propofol anesthesia in 10 healthy children undergoing elective urol. surgery. Anesthesia consisted of a steady-state propofol infusion and a continuous caudal epidural block. A transcranial Doppler probe was used to measure middle cerebral artery **blood flow** velocity. Randomization determined the sequence order of **N2O** (**N2O**/air or air/**N2O**) and end-tidal (ET) **CO2** concentration (25, 35, 45, and 55 mm Hg) using an exogenous source of **CO2**. At steady state, three sets of measurements of middle cerebral artery **blood flow** velocity, mean arterial **blood** pressure, and heart rate were recorded. A linear preservation of CCO2R was observed above 35 mm Hg of ETCO2, irresp. of **N2O**. A decrease in CCO2R to 1.4%-1.9% per mm of mercury was seen in the hypocapnic range (ETCO2 25-35 mm Hg) with both air and **N2O** . We conclude that **N2O** does not affect CCO2R during propofol anesthesia in children. When preservation of CCO2R is required, the combination of **N2O** with propofol anesthesia in children would seem suitable. The cerebral vasoconstriction caused by propofol would imply that hyperventilation to ETCO2 values less than 35 mm Hg may not be required because no further reduction in cerebral **blood flow** velocity would be achieved.
REFERENCE COUNT: 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 9 OF 181 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1983:69666 CAPLUS
DOCUMENT NUMBER: 98:69666
TITLE: Resorption of **carbon dioxide** and dinitrogen oxide (**N2O**) from newly formed

subcutaneous gas pockets
AUTHOR(S): Lagneaux, D.
CORPORATE SOURCE: Inst. Leon Fredericq Physiol. Hum. Norm., Univ. Liege, Liege, 4020, Belg.
SOURCE: Bulletin de la Societe Royale des Sciences de Liege (1982), 51(5-8), 287-315
CODEN: BSRSA6; ISSN: 0037-9565
DOCUMENT TYPE: Journal
LANGUAGE: French
AB The fundamental characteristics of gas exchange were studied in newly formed s.c. gas pockets in rat skin. The newly formed s.c. gas pockets are supplied by an autonomic vascular network, which is sensitive to the dilatory effect of **CO2**. This vasodilation causes an increase in the perfusion of the local vascular network and the opening of a greater number of capillaries, which increases the surface area in contact with gas and makes possible a more important resorption of gas. The efflux of **N2O**, a locally inert gas, is thus accentuated in the presence of excess **CO2**. When pCO2 is returned to its initial level, the efflux of **N2O** is subsequently identical to that observed when **N2O** is directly introduced into the s.c. pocket without excess **CO2**. The difference between the efflux of **CO2** and **N2O** depends not only on the possibility of **CO2** loading of the circulating **blood** but also on an increase in the **blood flow** which irrigates the s.c. pocket as a consequence of hypercapnic vasodilation.

L13 ANSWER 10 OF 181 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1999:274654 CAPLUS
DOCUMENT NUMBER: 131:56968
TITLE: Cortical NOS inhibition raises the lower limit of cerebral **blood flow**-arterial pressure autoregulation
AUTHOR(S): Jones, Stephen C.; Radinsky, Carol R.; Furlan, Anthony J.; Chyatte, Douglas; Perez-Trepichio, Alejandro D.
CORPORATE SOURCE: Department of Anesthesiology, Allegheny General Hospital, Pittsburgh, PA, 15212-4772, USA
SOURCE: American Journal of Physiology (1999), 276(4, Pt. 2), H1253-H1262
CODEN: AJPHAP; ISSN: 0002-9513
PUBLISHER: American Physiological Society
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The maintenance of constant cerebral **blood flow** (CBF) as arterial **blood** pressure is reduced, commonly referred to as CBF-pressure autoregulation, is typically characterized by a plateau until the vasodilatory capacity is exhausted at the lower limit, after which flow falls linearly with pressure. We investigated the effect of cortical, as opposed to systemic, nitric oxide synthase (NOS) inhibition on the lower limit of CBF-pressure autoregulation. Forty-four Sprague-Dawley rats were anesthetized with halothane and **N2O** in O2. With a closed cranial window placed the previous day in a ventilated and physiol. stable preparation, we determined the CBF using laser-Doppler flowmetry. Animals with low reactivity to inhaled **CO2** and suffused ADP or ACh were excluded. Five arterial pressures from 100 to 40 mmHg were obtained with controlled **hemorrhagic** hypotension under cortical suffusion with artificial cerebrospinal fluid (aCSF) and then again after suffusion for 35 (n = 5) and 105 min (n = 10) with aCSF, 10-3 M Nω-nitro-L-arginine (L-NNA; n = 12), or 10-3 M Nω-nitro-D-arginine (D-NNA; n = 5). An addnl. group (n = 7) was studied after a 105-min suffusion of L-NNA followed by a single **blood** withdrawal procedure. The lower limit of autoregulation was identified visually by four blinded reviewers as a change in the slope of the five-point plot of CBF vs. mean arterial **blood** pressure. The lower limit of 90 ± 4.3 mmHg after 105 min of 1 mM L-NNA suffusion was increased compared with the value in the time-control group of 75 ± 5.3 mmHg ($P < 0.01$; ANOVA) and the initial value of 67 ± 3.7 mmHg ($P < 0.001$). The lower limit of 84 ± 5.9 mmHg in seven animals with 105 min of suffusion of 1 mM L-NNA without previous **blood** withdrawal was significantly increased ($P < 0.01$) in comparison with 70 ± 1.9 mmHg

from those with just aCSF suffusion (n = 37). No changes in lower limit for the other agents or conditions, including 105 or 35 min of aCSF or 35 min of L-NNA suffusion, were detected. The lack of effect on the lower limit with D-NNA suffusion suggests an enzymic mechanism, and the lengthy L-NNA exposure of 105 min, but not 35 min, suggests inhibition of a diffusionally distant NOS source that mediates autoregulation. Thus, cortical suffusion of L-NNA raises the lower limit of autoregulation, strongly suggesting that nitric oxide is at least one of the vasodilators active during hypotension as arterial pressure is reduced from normal.
REFERENCE COUNT: 69 THERE ARE 69 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 11 OF 181 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1967:410116 CAPLUS
DOCUMENT NUMBER: 67:10116
TITLE: The effects of clinical concentrations of halothane on the **blood flow** and **oxygen** uptake of the cerebral cortex.
AUTHOR(S): McDowall, D. Gordon
CORPORATE SOURCE: Univ. Glasgow, Glasgow, UK
SOURCE: British Journal of Anaesthesia (1967), 39(3), 186-96
CODEN: BJANAD; ISSN: 0007-0912
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Clin. concns. of halothane vaporized in a mixture of **N2O** and O2 caused vasodilatation on the cerebral cortex of anesthetized dogs at constant arterial **CO2** tension. The vasodilatory action on the cerebral circulation was greater the higher the concns. of halothane. Two percent halothane increased **blood flow** through the cerebral cortex more than did 0.5% halothane. However, 4% halothane reduced mean **blood** pressure so markedly that **blood flow** was not elevated above the control value. The O uptake of the cerebral cortex was depressed by halothane, and this depression was greater with 2% halothane than with 0.5%. Halothane increased cerebral **blood flow** while reducing cerebral metabolic demand, resulting in an increase in the oxygenation of the brain.

L13 ANSWER 12 OF 181 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1961:66399 CAPLUS
DOCUMENT NUMBER: 55:66399
ORIGINAL REFERENCE NO.: 55:12654b-d
TITLE: Effects of hypercapnia on estimated hepatic **blood flow**, circulating splanchnic **blood** volume, and hepatic sulfobromophthalein clearance during general anesthesia in man
AUTHOR(S): Epstein, Robert M.; Wheeler, Henry O.; Frumin, M. Jack; Habif, David V.; Papper, Emanuel M.; Bradley, Stanley E.
CORPORATE SOURCE: Columbia Univ.
SOURCE: Journal of Clinical Investigation (1961), 40, 592-8
CODEN: JCINAO; ISSN: 0021-9738
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable
AB Estimated hepatic **blood flow**, splanchnic **blood** volume, and mean arterial pressure did not change significantly during light general anesthesia with thiopental and **N2O** following neuromuscular blockage with succinylcholine. Hypercapnia under these circumstances resulted in a significant increment in mean calculated vascular resistance. Sulfobromophthalein clearance and extraction by the liver, although unaffected by anesthesia, decreased significantly during hypercapnia. 24 references.

L13 ANSWER 13 OF 181 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1994:646075 CAPLUS
DOCUMENT NUMBER: 121:246075
TITLE: Effects of nitrous oxide on human regional cerebral **blood flow** and isolated pial arteries
AUTHOR(S): Reinstrup, Peter; Ryding, Erik; Algotsson, Lars;

Berntman, Leif; Uski, Tore
CORPORATE SOURCE: Gentofte Hospital, University Copenhagen, Hellerup, DK-2900, Den.
SOURCE: Anesthesiology (1994), 81(2), 396-402
CODEN: ANESAV; ISSN: 0003-3022
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Results from previous studies on the effect of nitrous oxide (**N2O**) on the cerebral circulation are conflicting. Early reports claim **N2O** to have no effect whereas recent findings demonstrate a cerebral cortical vasodilatation during **N2O** inhalation, but the regional cerebral **blood flow** (CBF) in the subcortical structures is unknown. Regional CBF was measured three-dimensionally with single photon emission computer-aided tomog. after injection of xenon 133 in 8 spontaneously breathing men (mean age 29.6 yr) during normocapnia and hypocapnia with and without inhalation of 50% **N2O**. 8 Isolated human pial arterial segments were mounted in organ baths. The segments were contracted with prostaglandin F2α and subjected to 30% **oxygen** and 5.6% **carbon dioxide** in nitrogen or **N2O**. Normocapnic young men had a global CBF of 55 mLΣ100 g-1 Σ min-1. Decreasing end-tidal **CO2** tension by 1.3 kPa (9.3 mmHg) reduced CBF uniformly, with a decrease in global CBF to 45 mLΣ100 g-1Σmin-1. During normocapnia, inhalation of 50% **N2O** increased mean CBF to 67 mLΣ100 g-1Σmin-1. Inhalation of 50% **N2O** during hypocapnia increased mean CBF to 63 mLΣ100 g-1Σmin-1. During **N2O** inhalation there was no significant difference in mean CBF between normo- and hypocapnia. However, during hypocapnia, but not during normocapnia, **N2O** inhalation significantly changed the distribution of regional CBF. Compared with hypocapnia without **N2O**, flow increased through the frontal (143%), parietal (140%) and temporal (133%) regions as well as through insula (151%), basal ganglia (145%) and thalamus (133%). In isolated human pial arteries, addition of **N2O** changed neither basal tension, nor the contraction elicited by prostaglandin F2α. Inhalation of 50% **N2O** increased global CBF mainly by augmenting flow in frontal brain structures. In contrast, changes in **carbon dioxide** without **N2O** affected CBF uniformly in the brain. The uneven change in distribution of the CBF when **N2O** was added during hypocapnia, the reduced **carbon dioxide** response, and the lack of effect of **N2O** on isolated human pial arteries suggest that **N2O** may increase metabolism in selected brain areas.

L13 ANSWER 14 OF 181 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1995:713536 CAPLUS
DOCUMENT NUMBER: 124:601
TITLE: The reducing effects of propofol/alfentanil vs. isoflurane on cerebral vascular reactivity to **carbon dioxide**
AUTHOR(S): Werner, C.; Standl, T.; Thiel, H.; Kochs, E.; Schulteam Esch, J.
CORPORATE SOURCE: Abt. Anaesthesiologie, Univ.-Krankenhaus Eppendorf, Hamburg, Germany
SOURCE: Anaesthesist (1995), 44(6), 417-22
CODEN: ANATAE; ISSN: 0003-2417
PUBLISHER: Springer
DOCUMENT TYPE: Journal
LANGUAGE: German
AB The effects of propofol (I)/alfentanil (II) vs. isoflurane (III) anesthesia on cerebral vascular reactivity to changes in **CO2** (IV) was compared transcranial Doppler sonog. (TCD). Seventeen ASA class I patients undergoing minor elective surgery were studied. In group 1, anesthesia was induced with thiopental (4 mg/kg) and II (15 g/kg). Endotracheal intubation was facilitated by vecuronium (V) (0.1 mg/kg). Anesthesia was maintained with 1% end-tidal III and **N2O** in O (6 L/min; FiO2 0.3). In group 2, anesthesia was induced with I (2 mg/kg), II (15 μ/kg), and V (0.1 mg/kg) for endotracheal intubation and maintained by infusion of I, II, and **N2O**-O2 according to the following protocol: I: 10, 8, and 6 mg/kg.h for 10 min each followed by 4 mg/kg.h;

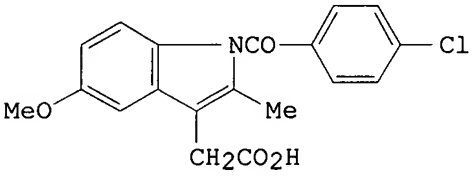
II: 55 μg/kg.h. Monitoring included measurement of mean arterial **blood** pressure (MAP, mm Hg), heart rate (HR), body temperature (T), endtidal IV (PetCO2, mm Hg), III concns., and arterial O2 saturation (SaO2, %). Mean **blood flow** velocity (Vmean, cm/s) was measured in the middle cerebral artery by a TCD system. Mech. ventilation was adjusted to achieve PetCO2 levels of 40-50-40-30 and 40 mm Hg. The IV reactivity index was calculated as ΔV mean/Δ PetCO2 (cm/s·mm Hg). MAP, HR, T, and SaO2 were constant over time and were not different between groups. The IV reactivity index over the IV range of 30-50 mm Hg was higher in III- (2.32 Δ cm/s·mm Hg) compared to I/II patients (1.15 Δ cm/s·mm Hg). The data show that although IV reactivity is maintained during both III and I/II anesthesia, the cerebral vascular response to IV was lower in I/II compared to III patients. These data suggest that IV reactivity is a function of the pre-existing cerebral vascular tone induced by the anesthetic.

L13 ANSWER 15 OF 181 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1981:117040 CAPLUS
DOCUMENT NUMBER: 94:117040
TITLE: A gas chromatographic technique for determination of **blood flow** and metabolism in individual organs (with special reference to the heart)
AUTHOR(S): Gisselsson, Lars; Ericsson, Martin; Johansson, Lennart
CORPORATE SOURCE: Dep. Anaesthesia, Malmo Gen. Hosp., Swed.
SOURCE: Upsala Journal of Medical Sciences (1980), 85(2), 113-24
CODEN: UJMSAP; ISSN: 0300-9734
DOCUMENT TYPE: Journal
LANGUAGE: English
AB A method for determining **blood flow**, O uptake, and **CO2** release in individual organs is presented. For **blood flow** measurements an inert gas (**N2O**) technique was used. **Blood** contents of O, **CO2**, and **N2O** were measured by gas chromatog. with the use of a special vacuum chamber for extracting the gases from **blood**. There was a strong correlation between the contents of O determined by the gas chromatog. and a spectrophotometric method. A good agreement was found between **CO2** in gas samples analyzed by the Scholander technique (1947) and by gas chromatog. A correlation coefficient of 0.998 was obtained between the **N2O** content calculated theor. and that determined by the gas chromatog. technique. The method makes it possible to simultaneously calculate **blood flow** in mL/100 g tissue/min, O uptake, and **CO2** production in an individual organ.

L13 ANSWER 16 OF 181 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1978:421160 CAPLUS
DOCUMENT NUMBER: 89:21160
TITLE: Cerebral **blood flow** and **oxygen** consumption in the newborn dog
AUTHOR(S): Hernandez, Milton J.; Brennan, Robert W.; Vannucci, Robert C.; Bowman, George S.
CORPORATE SOURCE: Milton S. Hershey Med. Cent., Pennsylvania State Univ., Hershey, PA, USA
SOURCE: American Journal of Physiology (1978), 234(5), R209-R215
CODEN: AJPHAP; ISSN: 0002-9513
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Cerebral **blood flow** (CBF), CBF responses to changes in arterial **CO2** tension, and cerebral metabolic rate for O (CMRO) were measured in newborn dogs, by a modification of the Kety and Schmidt technique employing 133Xe. Mongrel dogs of 1-7 days of age were paralyzed and passively ventilated with 70% **N2O** and 30% O. CBF was derived by anal. of paired serial 20-μL samples of arterial and cerebral venous **blood** from the superior sagittal sinus. At an arterial pCO2 of 36.9 torr and a mean arterial **blood** pressure of 62 torr, CBF was 23 mL/min/100 g. The arteriovenous O content difference

averaged 5.6 volume%, and CMRO was 1.13 mL O/min/100 g. CBF increased or decreased by 0.58 mL/min/100 g/torr change in pCO2. In the newborn, basal CBF and CBF responses to **CO2** are apparently considerably lower than those in the adult and parallel the lower metabolic needs of the newborn brain.

L13 ANSWER 17 OF 181 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1981:491110 CAPLUS
DOCUMENT NUMBER: 95:91110
TITLE: Effects of indomethacin on cerebral **blood flow** and **oxygen** consumption in barbiturate-anesthetized normocapnic and hypercapnic rats
AUTHOR(S): Dahlgren, Nils; Siesjo, Bo K.
CORPORATE SOURCE: Dep. Anasth., Univ. Lund, Lund, Swed.
SOURCE: Journal of Cerebral Blood Flow and Metabolism (1981), 1(1), 109-15
CODEN: JCBMDN; ISSN: 0271-678X
DOCUMENT TYPE: Journal
LANGUAGE: English
GI



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AB The effect of indomethacin (I) [53-86-1] was tested (10 mg/kg) on cerebral **blood flow**(CBF) and cerebral O consumption in rats anesthetized with 150 mg/kg of phenobarbital [50-06-6]. At normocapnia the barbiturate reduced CBF, measured with a 133Xe modification of the Kety-Schmidt technique, to about 50% of **N2O** control values as previously determined with a similar technique. At this CBF level, indomethacin induced a small, albeit highly significant decrease in CBF. Apparently, a reduction of this magnitude will escape detection with some CBF techniques in current use. Indomethacin induced a highly significant decrease in CBF during hypercapnia, demonstrating that the barbiturate does not eliminate the effect of indomethacin on **CO2** responsiveness. The magnitude of the reduction in **CO2** response was so large that it should be detected with most methods for measuring CBF. A comparison with previous data on animals under 70% **N2O** demonstrated that phenobarbital reduced the **CO2** responsiveness, defined as the ratio ΔCBF/ΔpCO2, to 39% of that observed under **N2O** anesthesia. With both types of anesthesia, indomethacin curtailed the **CO2** responsiveness 4- to 5-fold.

L13 ANSWER 18 OF 181 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1974:549657 CAPLUS
DOCUMENT NUMBER: 81:149657
TITLE: Cerebral **blood flow** measurements with xenon-133 during experimental metabolic alkalosis in the cat
AUTHOR(S): Pannier, J. L.
CORPORATE SOURCE: Lab. Norm. Pathol. Physiol., Univ. Ghent, Ghent, Belg.
SOURCE: Archives Internationales de Physiologie et de Biochimie (1974), 82(2), 413-15
CODEN: AIPBAY; ISSN: 0003-9799
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Cerebral **blood flow** was measured in anesthetized artificially ventilated cats under **N2O** anesthesia, paralyzed with gallamine, and the arterial pCO2 altered by replacing N2 by **CO2**, without change in the percent **N2O** and O2 in the inhaled mixture Infusions of Na2CO3 (0.23 or 0.30M) were made at a rate

sufficient to produce required changes in HCO3- concentration within 15 min and equilibria thereafter. NaCl infusions were used as electrolyte controls. Tissue clearance of 133Xe was done by external monitoring of the skull and cerebral **blood flow** calculated by the stochastic method. During 20-min infusion of Na2CO3, which raised the plasma HCO3- concentration by 14 mequiv/l., cerebral **blood flow** response to hypercapnia (5 or 7.5% **CO2** before and after infusion) was not affected. A 90-min infusion gave a smaller increase in plasma HCO3- concentration (12 mequiv/l.), reduced the cerebral **blood flow** response to hypercapnia from 115 to 93 ml/100 g/min, and increased the cerebrospinal fluid HCO3-concentration from 20.5 to 23.5 mequiv/l. More pronounced changes occurred when plasma HCO3- concentration was increased by 18 mequiv/l. for 90 min. Cerebral **blood flow** decreased from 130 to 80 ml/100/min, and cerebrospinal fluid HCO3- increased to 26.7 mequiv/l.

L13 ANSWER 19 OF 181 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1988:469668 CAPLUS
DOCUMENT NUMBER: 109:69668
TITLE: Separator membranes for mass spectrometry of **blood** gases: gas permeability with regard to the measurement of inert gases in the determination of organ **blood flows**
AUTHOR(S): Meyer, B. U.; Schroeter, W.; Zuechner, K.; Hellige, G.
CORPORATE SOURCE: Zent. - Physiol. Pathophysiol., Univ. Goettingen, Goettingen, D-3400, Fed. Rep. Ger.
SOURCE: Biomedizinische Technik (1988), 33(4), 66-72
CODEN: BMZTA7; ISSN: 0013-5585
DOCUMENT TYPE: Journal
LANGUAGE: German
AB Separator membranes used as a gas inlet for mass-spectrometric measurement of gas partial pressures in flowing **blood** or **blood** samples have an effect on the working characteristics of the analyzer. With the aim of optimizing such analyzing systems, the permeability of various membrane tubes and foils (polyethylene, polycarbonate, polyvinyl chloride, polyamide, silicone rubber, polytetrafluorethylene) for naturally occurring gases (N2, O2, **CO2**) and those used as indicator gases for the measurement of organ **blood flow** (He, Ne, Ar, **N2O**, Kr, Xe) was investigated. The main parameters of their permeation through these materials were: time constant of permeation (t63%), diffusion coefficient (D), solubility (β) and permeability (Pe). On the basis of the results, the suitability of the different membrane materials as optimal **blood** gas inlet in special applications of the inert gas techniques for the determining organ **blood flow**, is established.

L13 ANSWER 20 OF 181 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1978:416991 CAPLUS
DOCUMENT NUMBER: 89:16991
TITLE: Cerebrovascular **carbon dioxide** reactivity: role of a cholinergic mechanism modulated by anesthesia
AUTHOR(S): Scremin, O. U.; Rubinstein, E. H.; Sonnenschein, R. R.
CORPORATE SOURCE: Dep. Physiol., Univ. California Sch. Med., Los Angeles, CA, USA
SOURCE: Stroke (1978), 9(2), 160-5
CODEN: SJCCA7; ISSN: 0039-2499
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Cerebral cortical **blood flow** was measured in rabbits. The reactivity to **CO2** was highly dependent on the kind of anesthesia, being greatest under halothane [151-67-7] and least under **N2O**. Reactivity to **CO2** in halothane-anesthetized animals also depended on arterial **blood** pressure, being greatest when pressure was below 70 mm Hg. I.v. atropine blocked the increase in reactivity in halothane-anesthetized animals at low **blood** pressures. Conversely, i.v. eserine greatly increased the reactivity to **CO2** in **N2O**-anesthetized animals. Precollicular decerebration considerably decreased **CO2** reactivity of

halothane-anesthetized rabbits, whereas partial brain stem lesions that spared midline structures had no effect on **CO2** reactivity. Thus a central neurogenic mechanism with a cholinergic link may be responsible, at least in part, for the cerebrovascular effect of **CO2**. Moreover, the cerebrovascular effects of halothane may result from stimulation of the same system.

L13 ANSWER 21 OF 181 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1982:101871 CAPLUS
DOCUMENT NUMBER: 96:101871
TITLE: **Carbon dioxide** responses of the cerebral circulation during drug-induced hypotension in the cat
AUTHOR(S): Gregory, P.; Ishikawa, T.; McDowall, D. G.
CORPORATE SOURCE: Dep. Anaesth., Univ. Leeds, Leeds, LS2 9LN, UK
SOURCE: Journal of Cerebral Blood Flow and Metabolism (1981), 1(2), 195-201
CODEN: JCBMDN; ISSN: 0271-678X
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Hypotension was induced in cats with either practolol/trimetaphan or practolol/nitroprusside together with controlled **hemorrhage**. Arterial pCO2 was altered between 17 and 51 mm Hg by varying inspired **CO2** during constant-volume ventilation, first during control conditions of light halothane/**N2O** anesthesia and then during hypotension to mean **blood** pressure of 36-37 mm Hg. Cerebral cortical **blood flow** did not change with arterial pCO2 changes during trimetaphan hypotension, but some responsiveness to **CO2** persisted during nitroprusside hypotension, though at less than half control levels. No changes in pial artery diameter were seen with **CO2** during hypotension under either technique. It is postulated that **CO2** responsiveness persisted with nitroprusside because cerebral **blood flow** (CBF) values were higher when hypotension was produced with this drug than with trimetaphan. It would appear that hypocapnia does not further reduce CBF during trimetaphan hypotension but does do so with nitroprusside. However, the combination of hypocapnia and nitroprusside hypotension did not lower CBF below the values found during trimetaphan hypotension.

L13 ANSWER 22 OF 181 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1948:42539 CAPLUS
DOCUMENT NUMBER: 42:42539
ORIGINAL REFERENCE NO.: 42:8941h-i,8942a-b
TITLE: Cerebral **blood flow** and metabolism in schizophrenia. The effects of barbiturate seminarcosis, insulin coma, and electroshock
AUTHOR(S): Kety, S. S.; Woodford, R. B.; Harmel, M. H.; Freyhan, F. A.; Appel, K. E.; Schmidt, C. F.
CORPORATE SOURCE: Univ. of Pennsylvania, Philadelphia
SOURCE: Am. J. Psychiat. (1948), 104, 765-70
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable
AB Cerebral **blood flow** was measured by the **N2O** method (C.A. 40, 7274.8) in 22 schizophrenic patients. O2 and **CO2** arterio-venous differences were determined simultaneously. There was no deviation from the normal, as determined in 35 young males. It was concluded that there is no generalized change in the O2 utilization of the brain in schizophrenia. Seminarcosis, produced by Na pentothal or Na amytal in 8 patients, had no effect on cerebral **blood flow** or metabolism. During insulin hypoglycemia or coma in 7 schizophrenics there was a fall in O2 and glucose (I) arterio-venous differences, but the cerebral **blood flow** was well maintained. In deep insulin coma the I consumption fell 83% while the O2 consumption decreased only 45%. Utilization of the carbohydrate stores of the brain or of some other foodstuff was indicated. In 7 patients, 10 to 20 min. after electroshock convulsions, there was a moderate fall in cerebral O2 consumption, a marked decrease in cerebral **blood flow**, and a severe acidosis, as indicated by a sharp drop in **CO2** concentration and pH. The decreased **blood flow** can be

explained only partially by the change in **CO2** tension, and the decreased pH would tend to counteract even this effect.

L13 ANSWER 23 OF 181 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2000:302463 CAPLUS
DOCUMENT NUMBER: 133:663
TITLE: Nitrous oxide increases normocapnic cerebral **blood flow** velocity but does not affect the dynamic cerebrovascular response to step changes in end-tidal Pco2 in humans
AUTHOR(S): Aono, Mitsuo; Sato, Jiro; Nishino, Takashi
CORPORATE SOURCE: Department of Anesthesiology, Chiba University School of Medicine, Chiba, 260-8670, Japan
SOURCE: Anesthesia & Analgesia (Baltimore) (1999), 89(3), 684-689
CODEN: AACRAT; ISSN: 0003-2999
PUBLISHER: Lippincott Williams & Wilkins
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The authors sought to clarify the effect of **N2O** on the immediate responses of cerebral vasculature to sudden changes in arterial **CO2** tension in healthy humans. By use of a transcranial Doppler ultrasonog., **blood flow** velocity in the middle cerebral artery (VMCA) was measured during a step increase followed by a step decrease in end-tidal **CO2** tension (PETCO2) between normo- and hypercapnia while subjects inspired gas mixts. containing 70% O2 + 30% N2 (control) and 70% O2 + 30% **N2O** (**N2O**) sep. During the control condition, both step increase and decrease in PETCO2 produced rapid exponential changes in VMCA. An increase in VMCA produced by the step increase in PETCO2 was smaller and slower than a decrease in VMCA induced by the step decrease in PETCO2. These general features of the dynamic cerebrovascular response were not affected by substitution of **N2O** for N2 in the inspired gases although **N2O** increased baseline VMCA by 15% compared with the control condition. The authors conclude that **N2O** in itself does not affect the dynamic cerebrovascular response to arterial **CO2** changes, although it produces static mild cerebral vasodilation. This study suggests that **N2O** does not affect the dynamic cerebrovascular reactivity to acute arterial **CO2** changes, i.e., exponential changes in cerebral **blood flow** in response to step changes in alveolar **CO2** tension, although it does produce a mild increase in normocapnic cerebral **blood flow** velocity.
REFERENCE COUNT: 14 THERE ARE 14 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 24 OF 181 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1970:11194 CAPLUS
DOCUMENT NUMBER: 72:11194
TITLE: Effects of hexobendine[N,N'-dimethyl-N,N'-bis(3-(3',4',5'-trimethoxybenzoyloxy)-propyl)-ethylenediamine-2HCl] on cerebral **blood flow** and metabolism. Mechanism of action of cerebral vasodilatory drugs
AUTHOR(S): Kraupp, Otto; Nell, G.; Raberger, G.; Stuehlinger, W.
CORPORATE SOURCE: Inst. Pharmacol. Toxicol., Ruhr-Univ., Bochum, Fed. Rep. Ger.
SOURCE: Arzneimittel-Forschung (1969), 19(10), 1691-8
CODEN: ARZNAD; ISSN: 0004-4172
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The action of N,N'-dimethyl-N,N'-bis-[3-(3', 4',5' - trimethoxybenzoyloxy) - propyl] - ethylenediamine - 2HCl (hexobendine) (0.4 mg/kg i.v.) on the arterial levels and the cerebral arteriovenous differences of O, pCO2 ,H+, glucose, lactate, and pyruvate was studied in dogs anesthetized with morphine (3 mg/kg s.c.), chloralose (80 mg/kg i.v.) and a mixture (2:1) of **N2O** and O. The effects of hexabendine on the cerebral venous outflow (internal maxillary vein) was also studied under identical conditions on a sep. series of dogs. The difference in the O content between the femoral artery and the confluens sinium (AVDO2) decreased

transiently by .apprx.3.8-5.0 volume %, and the cerebral venous outflow increased by 30-40% following the administration of hexobendine. Although smaller, both effects were still apparent 30 min after the injection. Following the injection of hexobendine, the control cerebral arteriovenous difference (neg.) of H+ and pCO2 were more pos., due to the concomitant increase in **blood flow**. However, the allowance was made for the change in **blood flow**, the corrected cerebral arteriovenous difference values were significantly more neg. following the drug administration, indicative of an increased release of **CO2** and H+ from the brain. The control cerebral arteriovenous difference values for glucose were pos., and those for lactate and pyruvate were minimally neg. The O extraction ratio (OER) was +92% for glucose, -7.5% for lactate, and -0.8% for pyruvate. Within 1-2 min of the hexobendine injection, there was a highly significant increase in the OER for glucose to values of +125%, and the control values were not regained within 30 min. At the same time, the cerebral release of lactate sank transiently to zero. After the 7th min there was also a significant decrease in the cerebral release of pyruvate. This pattern was not in any way altered by making allowances for any simultaneous changes in the arterial levels of substrates. Based on these results a hypothesis was given in regard to a causal connection between pharmacodynamically induced changes in cerebral metabolism and **blood flow**.

L13 ANSWER 25 OF 181 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1965:492420 CAPLUS
DOCUMENT NUMBER: 63:92420
ORIGINAL REFERENCE NO.: 63:17000f-h
TITLE: The effect of different narcotic agents on local cerebral **blood flow** in cats
AUTHOR(S): Betz, E.; Oehmig, H.; Wuennenberg, W.
CORPORATE SOURCE: Univ. Marburg, Germany
SOURCE: Zeitschrift fuer Kreislaufforschung (1965), 54(5), 503-9
CODEN: ZEKRAW; ISSN: 0044-295X
DOCUMENT TYPE: Journal
LANGUAGE: German
AB The influence on local **blood flow** in the hypothalamus and thalamus was investigated with chronically implanted heat conductivity probes. Anesthesia with **N2O**, nembutal, and urethan caused slight decreases of local **blood flow** in the thalamus and hypothalamus; halothane increased the **blood flow**. In cats anesthetized with nembutal, the inhalation of 1.9% halothane decreased local cerebral **blood flow**. In **N2O** anesthesia the reactions of hypoxia and hypercapnia on local cerebral **blood flow** were similar to those in the conscious animals. When the cats were anesthetized with nembutal and urethan, hypoxia and hypercapnia raised cerebral **blood flow** in a lesser degree than in the conscious state. During haloethane anesthesia the high cerebral **blood flow** was reduced when the O content of the air was lowered to 8% O or the **CO2** content was raised to 8%. The arterial **blood** pressure decreased in each type of anesthesia, the greatest extent of **blood** pressure fall could be seen in halothane anesthesia.

L13 ANSWER 26 OF 181 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1991:17432 CAPLUS
DOCUMENT NUMBER: 114:17432
TITLE: Effects of nitrous oxide on global and regional cortical **blood flow**
AUTHOR(S): Deutsch, Georg; Samra, Satwant K.
CORPORATE SOURCE: Dep. Anesthesiol., Univ. Texas, Galveston, TX, USA
SOURCE: Stroke (1990), 21(9), 1293-8
CODEN: SJCCA7; ISSN: 0039-2499
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Regional cortical **blood flow** studies carried out by the 133Xe clearance technique on volunteers during the administration of 25% and 50% **N2O** and during baseline conditions (breathing room air or 100% O). Global cortical **blood flow** was higher

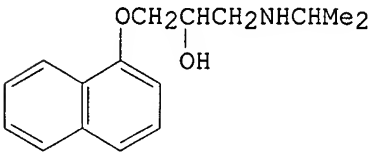
increased above baseline measures in all subjects by 50% **N2O** (mean increase 37% above the 100% O condition). A smaller but still significant increase was observed with 25% **N2O**. Anal. of regional cortical **blood flow** revealed heterogeneity in the pattern of changes, i.e., the baseline pattern was altered by the inhalation of **N2O** in a manner most often resulting in an accentuation of relative frontal **blood flow**. The anterior-posterior gradient in **N2O**-induced **blood flow** changes differed from that observed with simple vasodilator agents such as **CO2**, with which the increase is purely systemic and the baseline pattern is preserved. This indicates that **N2O** has differential effects on cerebral metabolism that may well reflect the typical alterations in experiential state reported by subjects.

L13 ANSWER 27 OF 181 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1985:89883 CAPLUS
DOCUMENT NUMBER: 102:89883
TITLE: Cerebrovascular aspects of converting-enzyme inhibition. I: effects of intravenous captopril in spontaneously hypertensive and normotensive rats
AUTHOR(S): Barry, David I.; Jarden, Jens O.; Paulson, Olaf B.; Graham, David I.; Strandgaard, Svend
CORPORATE SOURCE: Dep. Psychiatry, Rigshosp., Copenhagen, DK-2100, Den.
SOURCE: Journal of Hypertension (1984), 2(6), 589-97
CODEN: JOHYD3; ISSN: 0263-6352
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The cerebrovascular effects of angiotensin-converting enzyme [9015-82-1] inhibition were examined in normotensive and hypertensive rats. Cerebral **blood flow** was measured using the intracarotid 133Xe injection method in halothane/**N2O**-anesthetized animals. Following i.v. administration of captopril [62571-86-2] (10 mg/kg), cerebral **blood flow** autoregulation was markedly altered. Although cerebral **blood flow** was unchanged from baseline levels, both the lower and upper limits of autoregulation were reset to lower mean arterial pressure and the autoregulatory plateau was shortened. The lower limit was shifted 20-30 mm Hg, the upper limit 50-60 mm Hg, and the plateau shortened by 20-40 mm Hg. The effect resulted from compensatory autoregulatory constriction of small resistance vessels in the brain following captopril-induced dilatation of large resistance vessels. Thus, locally produced angiotensin II might play a role in the resistance of large cerebral arteries.

L13 ANSWER 28 OF 181 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1991:178231 CAPLUS
DOCUMENT NUMBER: 114:178231
TITLE: The effects of anesthetics and PaCO2 on the cerebrovascular, metabolic, and electroencephalographic responses to nitrous oxide in the rabbit
AUTHOR(S): Kaieda, Reiji; Todd, Michael M.; Warner, David S.
CORPORATE SOURCE: Coll. Med., Univ. Iowa, Iowa City, IA, USA
SOURCE: Anesthesia & Analgesia (Baltimore, MD, United States) (1989), 68(2), 135-43
CODEN: AACRAT; ISSN: 0003-2999
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The effects of nitrous oxide (**N2O**) on cerebral **blood flow** and metabolism, intracranial pressure (ICP), the EEG, etc., has been well described, at least when **N2O** is used alone. However, during neurosurgical procedures, **N2O** is almost always given in combination with either volatile or i.v. agents, and generally after the institution of some degree of hypocarbia. Unfortunately, the modifying influence of such interventions is not well known; therefore, the cerebral effects of 70% **N2O** were studied in rabbits anesthetized with either 1 Mac halothane or isoflurane, or with a fentanyl/pentobarbital combination, during both normocarbia (PaCO2 ≈ 40 mm Hg) and hypocarbia (PaCO2 ≈ 20 mm Hg). Cortical cerebral **blood flow** (CBFc) and sagittal sinus **blood flow**

(CBFss-as an index of global forebrain flow) were measured using the hydrogen clearance method. Cerebral **oxygen** consumption (CMRO2) was calculated, and intracranial pressure (ICP), central venous pressure, heart rate, mean arterial pressure, and the EEG were also recorded. CBFc during normocarbic halothane, isoflurane, and fentanyl-pentobarbital anesthesia was 69, 41, and 53 mL/100g/min, resp., with a significant difference between halothane and isoflurane. The addition of 70% **N2O** to all three anesthetics increased CBFc by 32%, 34%, and 36%, resp., during normocarbia and by 47%, 65%, and 27% during hypocarbia. A similar pattern was seen for CBFss. There were no significant differences in the response to **N2O** based either on anesthetic or PaCO2, except that the increase in CBFss produced by **N2O** during normocarbic fentanyl-pentobarbital anesthesia (10%) was less than that noted during normocarbic halothane anesthesia (39%). CMRO2 was not changed by addition of **N2O** regardless of anesthetic or PaCO2. **N2O** increased ICP under all conditions; the increments were small (about 1 mm Hg). The greatest EEG changes in responses to **N2O** were seen in halothane-anesthetized animals (a decrease in amplitude and increase in frequency). In contrast, no consistent EEG changes were seen with **N2O** in the fentanyl/pentobarbital group, while the only effect observed in the presence of isoflurane was the disappearance of the typical burst-suppression pattern. **N2O** increases CBF and ICP with each of the three background anesthetics during both normocarbia and hypocarbia. The magnitude of this response to **N2O** may be less during a fentanyl/barbiturate based anesthetic. These changes were not associated with alterations in CMRO2, suggesting that **N2O** may be a direct cerebral vasodilator in this species.

L13 ANSWER 29 OF 181 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1982:135557 CAPLUS
DOCUMENT NUMBER: 96:135557
TITLE: Effect of propranolol on local cerebral **blood flow** under normocapnic and hypercapnic conditions
AUTHOR(S): Dahlgren, Nils; Ingvar, Martin; Siesjo, Bo K.
CORPORATE SOURCE: Dep. Anesth., Univ. Lund, Lund, S-221 85, Swed.
SOURCE: Journal of Cerebral Blood Flow and Metabolism (1981), 1(4), 429-36
CODEN: JCBMDN; ISSN: 0271-678X
DOCUMENT TYPE: Journal
LANGUAGE: English
GI



I

AB The effect of dl-propranolol (dl-I) [13013-17-7] (2.5 mg/kg, i.v.) on local cerebral **blood flow** (CBF) in normocapnia was studied in rats maintained artificially ventilated on 70% **N2O** and 30% O. The method used was autoradiog. with [14C]iodoantipyrine. Although a single dose of propranolol, given 30 min prior to CBF measurements, somewhat reduced mean CBF values in all of the 22 structures analyzed, none of the changes were significant. The results confirm previous ones, in which overall CBF was measured, in showing that β-adrenergic mechanisms have little effect on normal cerebrovascular tone. Following a single dose of propranolol, results obtained in hypercapnia were equally neg.; neither did CBF fall significantly when propranolol was given by constant infusion during 15 min. Furthermore, local CBF did not differ between animals infused with dl-propranolol and d-propranolol (d-II) [5051-22-9]. Apparently, in the rat, propranolol has but small effects on the CBF response to hypercapnia, if any. Thus, local **CO2** responsiveness, calculated as ΔCBF/ΔPCO2,

varies with normocapnic flow rates.

L13 ANSWER 30 OF 181 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1986:400550 CAPLUS
DOCUMENT NUMBER: 105:550
TITLE: Isoflurane, halothane, and regional cerebral **blood flow** at various levels of PaCO2 in rabbits
AUTHOR(S): Scheller, Mark S.; Todd, Michael M.; Drummond, John C.
CORPORATE SOURCE: Dep. Anesthesiol., Univ. California, San Diego, La Jolla, CA, 92093, USA
SOURCE: Anesthesiology (1986), 64(5), 598-604
CODEN: ANESAV; ISSN: 0003-3022
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The effects of halothane [151-67-7] and isoflurane [26675-46-7] on regional cerebral **blood flow** (CBF) were studied in rabbits anesthetized with **N2O** and morphine at 3 different levels of PaCO2. Addition of 1 min, alveolar concentration (MAC) halothane to the background anesthetic caused flow to increase in all 3 regions studied (cortex, dorsal hippocampus, white matter) at all 3 levels of PaCO2. Addition of 1 MAC isoflurane to the background anesthetic caused CBF to significantly in all regions during hypocapnia. During normocapnia, CBF was unchanged with the addition of 1 MAC isoflurane in all regions and during hypercapnia, CBF increased only in the dorsal hippocampus following addition of 1 MAC isoflurane to the background anesthetic. Volatile anesthetic administration was associated with significant, although small, increases in intracranial pressure at all PaCO2, levels. Apparently, 1 MAC concns. of halothane and isoflurane have opposite effects on CBF when added to a **N2O**-morphine anesthetic during hypocapnia and that the effects of isoflurane on regional CBF are dependent on PaCO2 in rabbits under the anesthetic conditions of this experiment

=> d his

(FILE 'HOME' ENTERED AT 08:16:30 ON 20 MAR 2006)

FILE 'REGISTRY' ENTERED AT 08:16:38 ON 20 MAR 2006
L1 474 S ETHYL NITRATE
L2 52 S ETHYL NITRITE

FILE 'CAPLUS, MEDLINE, BIOSIS, EMBASE' ENTERED AT 08:17:46 ON 20 MAR 2006
L3 45910 S ETHYL NITRITE OR N2O OR NITROSYL ETHOXIDE OR NITROUS ETHER OR
L4 555 S 109-95-5/RN OR 8013-58-5/RN
L5 46026 S L4 OR L3

FILE 'REGISTRY' ENTERED AT 08:19:16 ON 20 MAR 2006
L6 1734 S CARBON DIOXIDE
L7 391 S ETHYL NITRITE OR N2O OR NITROSYL ETHOXIDE OR NITROUS ETHER OR

FILE 'CAPLUS, MEDLINE, BIOSIS, EMBASE' ENTERED AT 08:20:13 ON 20 MAR 2006
L8 738508 S CARBON DIOXIDE OR CO2 OR CARBON OXIDE OR CARBON-12 DIOXIDE OR
L9 9502 S L8 AND L5
L10 3065 S L9 AND (BLOOD OR HEM? OR HEMO? OR OXYGEN)
L11 290 S L10 AND (BLOOD FLOW)
L12 181 DUP REM L11 (109 DUPLICATES REMOVED)
L13 181 FOCUS L12 1-

=> s l11 and (liver or organ or intestine or kidney)
L14 26 L11 AND (LIVER OR ORGAN OR INTESTINE OR KIDNEY)

=> dup rem l14
PROCESSING COMPLETED FOR L14
L15 18 DUP REM L14 (8 DUPLICATES REMOVED)

=> focus
PROCESSING COMPLETED FOR L15
L16 18 FOCUS L15 1-

=> d ibib abs 1-18

L16 ANSWER 1 OF 18 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:117679 CAPLUS
DOCUMENT NUMBER: 138:163544
TITLE: Use of a **blood-flow** decrease preventing agent in conjunction with insufflating gas
INVENTOR(S): Stamler, Jonathan S.
PATENT ASSIGNEE(S): Duke University, USA
SOURCE: PCT Int. Appl., 16 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003011384	A1	20030213	WO 2002-US19810	20020712
W: AU, CA, JP				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR				
US 2003032917	A1	20030213	US 2001-919931	20010802
US 6676855	B2	20040113		
CA 2452529	AA	20030213	CA 2002-2452529	20020712
EP 1420851	A1	20040526	EP 2002-746630	20020712
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY, TR, BG, CZ, EE, SK				
JP 2004536878	T2	20041209	JP 2003-516614	20020712

PRIORITY APPLN. INFO.: US 2001-919931 A 20010802
WO 2002-US19810 W 20020712

AB A **blood-flow** decrease preventing agent is used to negate or reduce the decreased **oxygen** delivery in abdominal **organs** caused by insufflating gas. Preferably a gas is delivered into the abdominal cavity consisting essentially of the insufflating gas and the **blood-flow** decrease preventing agent. Very preferably, a gas is used consisting essentially of **carbon dioxide** as the insufflating gas and **Et nitrite** as the **blood-flow** to abdominal **organ** decrease preventing agent.

REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 2 OF 18 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1988:469668 CAPLUS
DOCUMENT NUMBER: 109:69668
TITLE: Separator membranes for mass spectrometry of **blood** gases: gas permeability with regard to the measurement of inert gases in the determination of **organ blood flows**
AUTHOR(S): Meyer, B. U.; Schroeter, W.; Zuechner, K.; Hellige, G.
CORPORATE SOURCE: Zent. - Physiol. Pathophysiol., Univ. Goettingen, Goettingen, D-3400, Fed. Rep. Ger.
SOURCE: Biomedizinische Technik (1988), 33(4), 66-72
CODEN: BMZTA7; ISSN: 0013-5585
DOCUMENT TYPE: Journal
LANGUAGE: German

AB Separator membranes used as a gas inlet for mass-spectrometric measurement of gas partial pressures in flowing **blood** or **blood** samples have an effect on the working characteristics of the analyzer. With the aim of optimizing such analyzing systems, the permeability of various membrane tubes and foils (polyethylene, polycarbonate, polyvinyl chloride, polyamide, silicone rubber, polytetrafluorethylene) for naturally occurring gases (N2, O2, **CO2**) and those used as indicator gases for the measurement of **organ blood flow** (He, Ne, Ar, **N2O**, Kr, Xe) was investigated. The main parameters of their permeation through these materials were: time constant of permeation (t63%), diffusion coefficient (D), solubility (β) and

permeability (Pe). On the basis of the results, the suitability of the different membrane materials as optimal **blood** gas inlet in special applications of the inert gas techniques for the determining **organ blood flow**, is established.

L16 ANSWER 3 OF 18 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1981:117040 CAPLUS
DOCUMENT NUMBER: 94:117040
TITLE: A gas chromatographic technique for determination of **blood flow** and metabolism in individual **organs** (with special reference to the heart)
AUTHOR(S): Gisselsson, Lars; Ericsson, Martin; Johansson, Lennart
CORPORATE SOURCE: Dep. Anaesthesia, Malmo Gen. Hosp., Swed.
SOURCE: Upsala Journal of Medical Sciences (1980), 85(2), 113-24
CODEN: UJMSAP; ISSN: 0300-9734
DOCUMENT TYPE: Journal
LANGUAGE: English
AB A method for determining **blood flow**, O uptake, and **CO2** release in individual **organs** is presented. For **blood flow** measurements an inert gas (**N2O**) technique was used. **Blood** contents of O, **CO2**, and **N2O** were measured by gas chromatog. with the use of a special vacuum chamber for extracting the gases from **blood**. There was a strong correlation between the contents of O determined by the gas chromatog. and a spectrophotometric method. A good agreement was found between **CO2** in gas samples analyzed by the Scholander technique (1947) and by gas chromatog. A correlation coefficient of 0.998 was obtained between the **N2O** content calculated theor. and that determined by the gas chromatog. technique. The method makes it possible to simultaneously calculate **blood flow** in mL/100 g tissue/min, O uptake, and **CO2** production in an individual **organ**.

L16 ANSWER 4 OF 18 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1961:66399 CAPLUS
DOCUMENT NUMBER: 55:66399
ORIGINAL REFERENCE NO.: 55:12654b-d
TITLE: Effects of hypercapnia on estimated hepatic **blood flow**, circulating splanchnic **blood** volume, and hepatic sulfobromophthalein clearance during general anesthesia in man
AUTHOR(S): Epstein, Robert M.; Wheeler, Henry O.; Frumin, M. Jack; Habif, David V.; Papper, Emanuel M.; Bradley, Stanley E.
CORPORATE SOURCE: Columbia Univ.
SOURCE: Journal of Clinical Investigation (1961), 40, 592-8
CODEN: JCINAO; ISSN: 0021-9738
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable
AB Estimated hepatic **blood flow**, splanchnic **blood** volume, and mean arterial pressure did not change significantly during light general anesthesia with thiopental and **N2O** following neuromuscular blockage with succinylcholine. Hypercapnia under these circumstances resulted in a significant increment in mean calculated vascular resistance. Sulfobromophthalein clearance and extraction by the **liver**, although unaffected by anesthesia, decreased significantly during hypercapnia. 24 references.

L16 ANSWER 5 OF 18 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1994:646075 CAPLUS
DOCUMENT NUMBER: 121:246075
TITLE: Effects of nitrous oxide on human regional cerebral **blood flow** and isolated pial arteries
AUTHOR(S): Reinstrup, Peter; Ryding, Erik; Algotsson, Lars; Berntman, Leif; Uski, Tore
CORPORATE SOURCE: Gentofte Hospital, University Copenhagen, Hellerup, DK-2900, Den.

SOURCE: Anesthesiology (1994), 81(2), 396-402
CODEN: ANESAV; ISSN: 0003-3022
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Results from previous studies on the effect of nitrous oxide (**N2O**) on the cerebral circulation are conflicting. Early reports claim **N2O** to have no effect whereas recent findings demonstrate a cerebral cortical vasodilatation during **N2O** inhalation, but the regional cerebral **blood flow** (CBF) in the subcortical structures is unknown. Regional CBF was measured three-dimensionally with single photon emission computer-aided tomog. after injection of xenon 133 in 8 spontaneously breathing men (mean age 29.6 yr) during normocapnia and hypocapnia with and without inhalation of 50% **N2O**. 8 Isolated human pial arterial segments were mounted in **organ** baths. The segments were contracted with prostaglandin F2α and subjected to 30% **oxygen** and 5.6% **carbon dioxide** in nitrogen or **N2O**. Normocapnic young men had a global CBF of 55 mLΣ100 g-1 Σ min-1. Decreasing end-tidal **CO2** tension by 1.3 kPa (9.3 mmHg) reduced CBF uniformly, with a decrease in global CBF to 45 mLΣ100 g-1Σmin-1. During normocapnia, inhalation of 50% **N2O** increased mean CBF to 67 mLΣ100 g-1Σmin-1. Inhalation of 50% **N2O** during hypocapnia increased mean CBF to 63 mLΣ100 g-1Σmin-1. During **N2O** inhalation there was no significant difference in mean CBF between normo- and hypocapnia. However, during hypocapnia, but not during normocapnia, **N2O** inhalation significantly changed the distribution of regional CBF. Compared with hypocapnia without **N2O**, flow increased through the frontal (143%), parietal (140%) and temporal (133%) regions as well as through insula (151%), basal ganglia (145%) and thalamus (133%). In isolated human pial arteries, addition of **N2O** changed neither basal tension, nor the contraction elicited by prostaglandin F2α. Inhalation of 50% **N2O** increased global CBF mainly by augmenting flow in frontal brain structures. In contrast, changes in **carbon dioxide** without **N2O** affected CBF uniformly in the brain. The uneven change in distribution of the CBF when **N2O** was added during hypocapnia, the reduced **carbon dioxide** response, and the lack of effect of **N2O** on isolated human pial arteries suggest that **N2O** may increase metabolism in selected brain areas.

L16 ANSWER 6 OF 18 MEDLINE on STN
ACCESSION NUMBER: 2005057215 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15650635
TITLE: A method to attenuate pneumoperitoneum-induced reductions in splanchnic **blood flow**.
AUTHOR: Ali Nishath Athar; Eubanks W Steve; Stamler Jonathan S; Gow Andrew J; Lagoo-Deenadayalan Sandhya A; Villegas Leonardo; El-Moalem Habib E; Reynolds James D
CORPORATE SOURCE: Department of Anesthesiology, Duke University Medical Center, Durham, North Carolina 27710, USA.
CONTRACT NUMBER: HD042471 (NICHD)
SOURCE: Annals of surgery, (2005 Feb) Vol. 241, No. 2, pp. 256-61.
Journal code: 0372354. ISSN: 0003-4932.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200502
ENTRY DATE: Entered STN: 20050203
Last Updated on STN: 20050223
Entered Medline: 20050222

AB OBJECTIVE: To determine if increasing nitric oxide bioactivity by inclusion of **ethyl nitrite** (ENO) in the insufflation admixture would attenuate pneumoperitoneum-induced decreases in splanchnic perfusion. SUMMARY BACKGROUND DATA: **Organ blood flow** is reduced during pneumoperitoneum and can contribute to laparoscopy-associated morbidity and mortality. Previous attempts to control such decreases in flow have been ineffective. METHODS: Laser-Doppler flow probes were placed on the **liver** and right

kidney of anesthetized pigs. After a baseline recording period, animals were insufflated to a final intraperitoneal pressure of 15 mm Hg. Group one received **CO2** (standard practice), whereas group 2 received **CO2** plus 100 ppm ENO. Insufflation was maintained for 60 minutes and then the abdomen was manually deflated; monitoring was continued for another 60 minutes. RESULTS: **CO2** insufflation (n = 5) cut **liver blood flow** in half; **liver** flow remained at this level throughout the postinsufflation period. Inclusion of 100 ppm ENO (n = 6) attenuated both the acute and prolonged **blood flow** decreases. Statistical modeling of the data showed that, on average, **liver blood flow** was 14.3 U/min higher in the ENO pigs compared with the **CO2** group (P = 0.0454). In contrast, neither treatment significantly altered **kidney blood flow** (P = 0.6215). CONCLUSION: The data indicate that ENO can effectively attenuate pneumoperitoneum-induced **blood flow** decreases within the peritoneal cavity. The result suggests a novel therapeutic method of regulating **hemodynamic** changes during laparoscopic procedures.

L16 ANSWER 7 OF 18 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2004:93978 BIOSIS
DOCUMENT NUMBER: PREV200400095530
TITLE: Use of a **blood-flow** decrease preventing agent in conjunction with insufflating gas.
AUTHOR(S): Stamler, Jonathan S. [Inventor, Reprint Author]
CORPORATE SOURCE: ASSIGNEE: Duke University
PATENT INFORMATION: US 6676855 20040113
SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (Jan 13 2004) Vol. 1278, No. 2. http://www.uspto.gov/web/menu/patdata.html. e-file. ISSN: 0098-1133 (ISSN print).
DOCUMENT TYPE: Patent
LANGUAGE: English
ENTRY DATE: Entered STN: 11 Feb 2004
Last Updated on STN: 11 Feb 2004
AB A **blood-flow** decrease preventing agent is used to negate or reduce the decreased **oxygen** delivery in abdominal **organs** caused by insufflating gas. Preferably a gas is delivered into the abdominal cavity consisting essentially of the insufflating gas and the **blood-flow** decrease preventing agent. Very preferably, a gas is used consisting essentially of **carbon dioxide** as the insufflating gas and **ethyl nitrite** as the **blood-flow** to abdominal **organ** decrease preventing agent.

L16 ANSWER 8 OF 18 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 1982:285717 BIOSIS
DOCUMENT NUMBER: PREV198274058197; BA74:58197
TITLE: SYSTEMIC AND REGIONAL **BLOOD FLOW** DISTRIBUTION IN UNANESTHETIZED SWINE AND SWINE ANESTHETIZED WITH HALOTHANE PLUS NITROUS OXIDE HALOTHANE OR ENFLURANE.
AUTHOR(S): TRANQUILLI W J [Reprint author]; MANOHAR M; PARKS C M; THURMON J C; THEODORAKIS M C; BENSON G J
CORPORATE SOURCE: 222 LARGE ANIM CLIN, DEP VET BIOSCI, COLL VET MED, UNIV ILLINOIS-URBANA, URBANA, ILL 61801, USA
SOURCE: Anesthesiology (Hagerstown), (1982) Vol. 56, No. 5, pp. 369-379. CODEN: ANESAV. ISSN: 0003-3022.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH
AB To study the distribution of cardiac output during various anesthetic regimens, regional **organ blood flow** was measured using 15-μm-diameter radionuclide-labeled microspheres injected into the left atrium in 9 pigs during resting unanesthetized state (control), halothane (inspired concentration = 1.25%) + **N2O** (50%), halothane (inspired concentration = 2.25%), and enflurane (inspired concentration = 4.0%) anesthesia. The order of the last 2 treatments [halothane (2.25%) and enflurane (4.0%)] was randomized among the 9 pigs.

All anesthetic steps employed intermittent positive-pressure ventilation to maintain PaCO2 [arterial partial pressure of **CO2**] close to control values. Animals were allowed to recover towards the control state before changing to the next anesthetic regimen. Forty-five minutes were allowed for equilibration with each anesthetic regimen before **hemodynamic** measurements were made. Cardiac output and mean arterial **blood** pressure decreased significantly from control values with each of the 3 anesthetized steps. The decrease in cardiac output was greatest with halothane and least with halothane + **N2O** . **Blood flow** per unit weight of the cardiac, renal and splanchnic tissues decreased significantly with each anesthetic regimen, whereas brain **blood flow** and hepatic arterial **blood flow** were unaltered from control values. The percent cardiac output received by the brain increased with halothane (119%) and enflurane (102%) anesthesia, while it was unaltered for the heart, renal and splanchnic **organs**. Percentage of total cardiac output received by **liver** via the hepatic artery increased by 162% during halothane and 133% during enflurane, when compared to control values. During halothane + **N2O** anesthesia, the percent of cardiac output going to the brain was not increased significantly. Evidently, cardiac output and individual **organ**/tissue **blood flow** was better maintained during halothane + N20 than halothane or enflurane anesthesia.

L16 ANSWER 9 OF 18 EMBASE

ACCESSION NUMBER: 75054835 EMBASE
DOCUMENT NUMBER: 1975054835
TITLE: Hepatic **blood flow** and cardiac output during halothane anesthesia: an animal study.
AUTHOR: Juhl B.; Einer Jensen N.
CORPORATE SOURCE: Lab. Clin. Physiol., Odense Univ. Hosp., Odense
SOURCE: Acta Anaesthesiologica Scandinavica, (1974) Vol. 18, No. 2, pp. 114-122. .
CODEN: AANEAB
DOCUMENT TYPE: Journal
FILE SEGMENT: 037 Drug Literature Index
024 Anesthesiology
030 Pharmacology
023 Nuclear Medicine
018 Cardiovascular Diseases and Cardiovascular Surgery
LANGUAGE: English

AB Correlated values of cardiac output and **liver blood flow** were measured on eight dogs. Measurements were carried out under basis anesthesia with O2 **N2O** barbiturate gallamonium with controlled ventilation as well as during the addition of 0.5% and 1.0% halothane to the inspiratory air. During the latter type anesthesia, a parallel fall in the cardiac output and the **liver blood flow** was demonstrated. With 0.5% halothane, the fall was on the average 30% and 25% of the initial values respectively, and with 1.0% halothane it was 49% and 45% of the initial values. The individual changes were extremely dependent upon simultaneous changes in PaCO2. The fall in cardiac output and **liver blood flow** was least when the PaCO2 increased at the same time, and greatest when the PaCO2 fell simultaneously. No changes were demonstrated in the total peripheral resistance and the resistance in the splanchnic region. It is concluded that changes in the cardiac output and hepatic **blood flow** brought about by the PaCO2 are parallel, and this appears to be undiminished during anesthesia with O2 **N2O** halothane in the concentrations used in this study.

L16 ANSWER 10 OF 18 EMBASE

ACCESSION NUMBER: 85194488 EMBASE
DOCUMENT NUMBER: 1985194488
TITLE: [Simultaneous mass spectrometric partial pressure measurement of **CO2** and **N2O** in **blood** for metabolic investigations and **blood flow** measurements].

GLEICHZEITIGE MASSENSPEKTROMETRISCHE PARTIALDRUCKMESSUNG IN BLUT VON **CO2** UND **N2O** FUR STOFFWECHSELUNTERSUCHUNGEN UND DURCHBLUTUNGSMESSUNGEN.

AUTHOR: Beste K.W.
CORPORATE SOURCE: Medizinische Hochschule Hannover, Abteilung Physikalische Medizin und Rehabilitation, D-3000 Hannover 61, Germany

SOURCE: Biomedizinische Technik, (1985) Vol. 30, No. 7-8, pp. 192-196. .
CODEN: BMZTA7
COUNTRY: Germany
DOCUMENT TYPE: Journal
FILE SEGMENT: 027 Biophysics, Bioengineering and Medical Instrumentation
002 Physiology
LANGUAGE: German
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 911210
Last Updated on STN: 911210

DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

ACCESSION NUMBER: 78254664 EMBASE
DOCUMENT NUMBER: 1978254664

TITLE: The effect on the splanchnic **blood flow** and cardiac output of various **carbon dioxide** concentrations during fluroxene (Fluoromar) anaesthesia.

AUTHOR: Juhl B.; Einer Jensen N.
CORPORATE SOURCE: Inst. Anaesthesiol., Odense Univ., Odense, Denmark

SOURCE: Acta Anaesthesiologica Scandinavica, (1977) Vol. 21, No. 6, pp. 449-456. .
CODEN: AANEAB
COUNTRY: Denmark
DOCUMENT TYPE: Journal
FILE SEGMENT: 037 Drug Literature Index
024 Anesthesiology
030 Pharmacology
LANGUAGE: English

AB Seven dogs premedicated with pethidine 10 mg/kg body weight, were anaesthetized with mebumal natrium 25 mg/kg body weight i.v. and gallamoni jodidum 80 mg, together with O2 - **N2O** in a ratio of 1 to 1. Thereafter four dogs were constantly hyperventilated and three constantly hypoventilated under stepwise increasing anaesthesia with fluoromar® (=fluroxene), up to 6% inspiratory concentration. A change was made between the groups at this fluoromar concentration, from hyper, to hypoventilation and vice versa, after which the fluoromar concentration was reduced stepwise to 0% inspiratory. During the course of this anaesthesia, the related values for flow, pressure and resistance in the sytemic and splanchnic circulations were measured at fluoromar concentrations: 0-1 1/2-3-6-6-3-1 1/2-0%. The relative changes in the systemic circulation during increasing concentrations of fluoromar are independent of hypo- of hypercapnia, while the absolute magnitudes of the measured parameters are strongly dependent on the **CO2** tensions. The hypercapnic dogs had the highest cardiac output, stroke volume and most rapid pulse, as well as the lowest peripheral resistance. The same group of dogs had the lowest **liver blood flow** and the greatest splanchnic resistance. The mean pressures in the aorta, right atrium and portal vein were not different between the groups. A straight line dependence at 6% fluoromar, was demonstrated between cardiac index and Pa(**CO2**):cardiac index (l.min-1.m-2) = 1.22 + 0.23 x Pa(**CO2**) (kPa), ((cardiac index (l.min-1.m-2) = 1.22 + 0.03 x Pa(**CO2**) (mmHg)), and peripheral resistance and Pa(**CO2**):peripheral resistance (kPa 1-1 min) = 12.85 - 0.5 x Pa(**CO2**) (kPa), ((peripheral resistance (dyn.sec.cm-5 x 10-3) = 7.72-0.04xPa(**CO2**) (mmHg)). The pressure in the portal vein appears to rise and the **liver** flow to fall with rising **carbon dioxide** tension, but the relationship is not significant. In the latter half of the investigation only small changes were demonstrated

which could be related to the falling concentration of fluoromar. These were slight rises in the mean pressure of the aorta and cardiac frequency, similar for both groups. In addition, a slight rise in the peripheral resistance and splanchnic resistance was observed in the hypocapnic group. No changes were seen in the cardiac output and **liver** flow in any of the groups during falling fluoromar concentrations. The absolute magnitude of the measured parameters in relation to the **CO2** tension reflects the conditions in the first half of the investigation, but the difference between the groups is less pronounced. This was partly due to the smaller difference in Pa(**CO2**) between the groups in this part of the investigation and partly due to the time and experimental course.

L16 ANSWER 12 OF 18 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 1976:204827 BIOSIS
DOCUMENT NUMBER: PREV197662034827; BA62:34827
TITLE: EXPERIMENTAL DETERMINATION OF **BLOOD** ALCOHOL METABOLISM WITH DOGS IN STANDARDIZED **HEMORRHAGIC** SHOCK.
AUTHOR(S): KAUFMANN H; TAUSCH D; HARBAUER G; WAGNER H-J
SOURCE: Zeitschrift fuer Rechtsmedizin, (1976) Vol. 77, No. 2, pp. 79-89.
CODEN: ZRMDAN. ISSN: 0044-3433.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: Unavailable
AB Ethanol at a dosage of 3 g/kg reduced body weight was injected i.v. into mongrel dogs resulting in a **blood** alcohol concentration of approximately 2.9 mg/ml. One hour after injection the dogs were anesthetized with halothane-**N2O**/O2 and **blood** was withdrawn until the **blood** pressure was reduced to 40 mm Hg. This usually required removal of about 30-40% of the total **blood** volume. The resulting **hemorrhagic** shock was ascertained by monitoring **blood** pH, PCO2 [partial **CO2** pressure], PO2 [partial O2 pressure], lactate, pyruvate and **blood** electrolytes. A **blood** specimen for enzymatic alcohol determination (ADH [alcohol dehydrogenase test]) was obtained every 30 min over a period of 3 h. Compared with equally dosed controls the dogs in **hemorrhagic** shock showed a significant (P = 0.005) reduction of the **blood** alcohol decay rate which is explained by the diminished **blood flow** through the **liver** and the hypoxemic metabolic situation in shock.

L16 ANSWER 13 OF 18 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 1982:239782 BIOSIS
DOCUMENT NUMBER: PREV198274012262; BA74:12262
TITLE: EFFECTS OF TRIMETHAPHAN NITROPRUSSIDE AND NITRO GLYCERIN ON **ORGAN HEMODYNAMICS**.
AUTHOR(S): NAKAGAWA M [Reprint author]
CORPORATE SOURCE: ICU, TOKYO MED AND DENTAL UNIV, TOKYO, 113 JAPAN
SOURCE: Japanese Journal of Anesthesiology, (1981) Vol. 30, No. 12, pp. 1301-1309.
CODEN: MASUAC. ISSN: 0021-4892.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: JAPANESE
AB Adult mongrel dogs [8] were anesthetized with secobarbital, **N2O** and O2, and the changes in **organ hemodynamics** induced by trimethaphan (TM), nitroprusside (SNP) and nitroglycerine (TNG) were examined. Injected doses were 5 .apprx. 20 μg/kg per min of TM, 10 .apprx. 50 μg/kg per min of SNP and10 .apprx. 50 μg/kg per min of TNG, which were larger than clinical dosage. Decrease in systolic **blood** pressure was 34.2 ± 7.1% with SNP and 19.7 ± 8.6% with TNG. The effect of TNG on **blood** pressure was moderate and difficult to regulate. Cardiac output (CO) increased with SNP, but decreased with TM and TNG. These changes were not significant, and the 3 drugs can be used safely to induce hypotensive anesthesia. Carotid

blood flow was increased with each drug. Brain **blood flow** was well sustained. Renal **blood flow** was decreased 31% with TM, but SNP and TNG caused little change. TM increased PaCO2 [arterial partial **CO2** pressure] and decraesed pH, and SNP decreased PaO2 [arterial partial O2 pressure] slightly.

L16 ANSWER 14 OF 18 MEDLINE on STN
ACCESSION NUMBER: 89285464 MEDLINE
DOCUMENT NUMBER: PubMed ID: 2500033
TITLE: Regional cerebral **blood flow** and glucose utilization during hypoglycemia in newborn dogs.
AUTHOR: Mujsce D J; Christensen M A; Vannucci R C
CORPORATE SOURCE: Department of Pediatrics, Pediatric Neurology and Neonatology, Milton S. Hershey Medical Center, Pennsylvania State University, Hershey 17033.
CONTRACT NUMBER: HD-15738 (NICHD)
SOURCE: The American journal of physiology, (1989 Jun) Vol. 256, No. 6 Pt 2, pp. H1659-66.
Journal code: 0370511. ISSN: 0002-9513.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198907
ENTRY DATE: Entered STN: 19900309
Last Updated on STN: 19970203
Entered Medline: 19890718

AB To assess alterations in regional cerebral **blood flow** and glucose utilization during perinatal hypoglycemia, newborn dogs (2-7 days postnatal age) were anesthetized with halothane, tracheostomized, paralyzed, and artificially ventilated with 70% **N2O**-30% O2 to maintain arterial normoxia and normocapnia (arterial PO2 greater than 60 mmHg; arterial PCO2: 35-42 mmHg; arterial pH: 7.35-7.45). Regional cerebral **blood flow** (rCBF) and glucose utilization (rCGU) were determined with iodo-[14C]antipyrine and 2-deoxy-[14C]glucose as the radioactive tracers, respectively. Hypoglycemia with **blood** glucose concentrations averaging 0.9 mmol/l was achieved within 90-120 min in 10 animals using intermittent intravenous injections of regular insulin; 10 control animals received 0.9% saline (**blood** glucose = 9 mmol/l). During hypoglycemia, mean arterial **blood** pressure was 81% of control, whereas heart rate was unchanged. Arterial O2 and acid-base balance were well maintained (arterial PO2 = 68 mmHg; PCO2 = 37 mmHg; pH = 7.35). Hypoglycemia was associated with significant increases in rCBF in all of 16 analyzed structures, ranging from 172% (parietal white matter) to 249% (thalamus) of control values (17-65 ml.100 g-1.min-1). During hypoglycemia, rCGU was relatively unchanged from normoglycemic values in 11 of 16 brain structures. Significant reductions in rCGU were seen only in occipital white matter (-31%) and in the cerebellar vermis and **hemisphere** (-31 and -43%, respectively). CGU actually increased slightly in the pons and medulla (+12 and +19%, respectively).(ABSTRACT TRUNCATED AT 250 WORDS)

L16 ANSWER 15 OF 18 EMBASE
ACCESSION NUMBER: 83016183 EMBASE
DOCUMENT NUMBER: 1983016183
TITLE: Cardiovascular and biochemical changes in dogs during etomidate-nitrous oxide anaesthesia.
AUTHOR: Van Lambalgen A.A.; Bronsveld W.; Van Den Bos G.C.; et al.
CORPORATE SOURCE: Physiol. Lab., Free Univ., 1081 BT Amsterdam, Netherlands
SOURCE: Cardiovascular Research, (1982) Vol. 16, No. 10, pp. 599-606. .
CODEN: CVREAU
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal
FILE SEGMENT: 037 Drug Literature Index
018 Cardiovascular Diseases and Cardiovascular Surgery
LANGUAGE: English

ENTRY DATE: Entered STN: 911209
Last Updated on STN: 911209

AB Etomidate was infused in dogs to test whether the drug could be used for long-lasting stable anaesthesia. In six dogs anaesthesia was induced with pentothal (20 mg.kg-1) and continued with a constant infusion of etomidate (4 mg.kg-1.h-1). The animals were ventilated with a mixture of O2 and **N2O** (1:2). Several haemodynamic (heart rate, arterial pressure, central venous pressure, pulmonary artery pressure, left ventricular end-diastolic pressure, dP/dt[LV], cardiac output, systemic vascular resistance) and biochemical (pO2, pCO2, pH, O2 content, lactate, glucose) variables together with the distribution of cardiac output (radioactive microsphere method) were studied for four and a half hours. In the course of the experiment cardiac output (thermodilution), stroke volume and dP/dt[LV] (catheter tip manometer) decreased by 38, 40 and 25% respectively while systemic vascular resistance increased (38%); the pH in mixed venous **blood** fell from 7.31 to 7.26. **Blood flow** to gastrointestinal tract, pancreas, skin and muscle decreased in proportion with the fall in cardiac output. Myocardial, renal, adrenal and splenic **blood flow** did not change. Hepatic artery flow decreased (67%). **Liver** perfusion was thus severely affected since portal **blood flow** also decreased. **Oxygen** consumption in the systemic, myocardial and splanchnic bed was not affected. In four other dogs **blood** etomidate levels were also measured. They rose gradually in the course of the experiment even though concentration and infusion rate of etomidate were kept constant throughout the four and a half hour experiment in these and all other dogs. Etomidate thus caused cardiovascular depression when it was used as long-lasting anaesthetic. Insufficient clearance of etomidate by the **liver** through deficient hepatic **blood flow** may be a causative factor.

L16 ANSWER 16 OF 18 MEDLINE on STN
ACCESSION NUMBER: 95262184 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7743571
TITLE: Noninvasive ICG clearance test for estimating hepatic **blood flow** during halothane and isoflurane anaesthesia.
AUTHOR: Kanaya N; Iwasaki H; Namiki A
CORPORATE SOURCE: Department of Anesthesiology, School of Medicine, Sapporo Medical University, Japan.
SOURCE: Canadian journal of anaesthesia = Journal canadien d'anesthesie, (1995 Mar) Vol. 42, No. 3, pp. 209-12. Journal code: 8701709. ISSN: 0832-610X.
PUB. COUNTRY: Canada
DOCUMENT TYPE: (CLINICAL TRIAL) Journal; Article; (JOURNAL ARTICLE) (RANDOMIZED CONTROLLED TRIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199506
ENTRY DATE: Entered STN: 19950621
Last Updated on STN: 19980206
Entered Medline: 19950615

AB The purpose of this study was to estimate hepatic **blood flow** during halothane (HAL) or isoflurane (ISO) anaesthesia with the noninvasive indocyanine green (ICG) clearance test. Twenty-four ASA status I adult patients, scheduled for elective surgery without **liver** disease, were allocated into four groups. Before surgery, ICG clearance was measured twice in patients before and after 1, 2 MAC HAL-**N2O**, 1 and 2 MAC ISO-**N2O** anaesthesia by ICG clearance meter (Sumitomo Electronics, Japan). This method eliminates the **blood** sampling and delay of the conventional ICG test. The ICG clearance is displayed in two indices: K (ICG disappearance rate) and R15 (ICG retention rate 15 min after 0.5 mg.kg-1 ICG injection). Indirect **blood** pressure and heart rate were measured simultaneously. Mean arterial pressure (MAP) decreased by 27, 22 and 29% with 2 MAC HAL-**N2O**, and with 1 and 2 MAC ISO-**N2O** groups, respectively ($P < 0.05$). The ICG clearance was less ($P < 0.05$) in the 2 MAC HAL-**N2O** group (K, 0.087 min-1) than with either awake (K, 0.131 min-1)

or other groups (K, 0.114 and 0.144 min-1, in the 1 MAC HAL and ISO-**N2O** groups, respectively) in spite of a similar degree of the depletion of MAP in 2 MAC ISO-**N2O** group (K, 0.124 min-1). We conclude that isoflurane has a more favourable effect on **liver** circulation than does halothane.

L16 ANSWER 17 OF 18 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 77202709 EMBASE
DOCUMENT NUMBER: 1977202709
TITLE: The effect of hypocapnia on systemic and regional **blood flows** during neuroleptanesthesia in dogs.
AUTHOR: In Nami H.
CORPORATE SOURCE: Dept. Anesthesiol., Tokyo Univ. Fac. Med., Tokyo, Japan
SOURCE: Japanese Journal of Anesthesiology, (1976) Vol. 25, No. 11, pp. No.94. .
CODEN: MASUAC
DOCUMENT TYPE: Journal
FILE SEGMENT: 037 Drug Literature Index
024 Anesthesiology
LANGUAGE: English

AB The effect of hypocapnia on cardiac output and its distribution to individual **organs** were investigated in dogs under neuroleptanesthesia, using radioactive microspheres. 16 dogs were used. Anesthesia was induced with thalamonal, and was maintained with **N2O** and fentanyl. Ventilation was controlled with a ventilator. The tidal volume (27 ml/kg) and frequency (20/min) were kept constant throughout the study. 2 consecutive stages of Pa(**CO2**) levels (normo- and hypocapnia) were induced in random sequence in each dog by changing the inspired **CO2** concentration. After maintaining each Pa(**CO2**) level for 30 min, cardiac output was measured and 50 micron radioactive microspheres were injected into the left ventricle. The fractional distribution of cardiac output to individual areas was calculated by the method of Rudolph and Heymann. Mean values of Pa(**CO2**) at normo- and hypocapnia were around 40 and 20 mmHg, respectively. Hypocapnia produced a significant reduction in cardiac output (-20%) and a change in the distribution of **blood flow** to regional areas. The fraction of cardiac output to the brain decreased significantly, while that to the **kidney** and skeletal muscle increased significantly. The fraction of cardiac output to the other areas remained unchanged.

L16 ANSWER 18 OF 18 MEDLINE on STN

ACCESSION NUMBER: 83174461 MEDLINE
DOCUMENT NUMBER: PubMed ID: 6404083
TITLE: Lysine-vasopressin in excisional treatment of burns in pigs. Decreased **blood** loss and earlier circulatory recovery.
AUTHOR: Vernersson E; Ahlgren I; Aronsen K F; Palmer B
SOURCE: Acta chirurgica Scandinavica, (1983) Vol. 149, No. 1, pp. 15-22.
Journal code: 7906530. ISSN: 0001-5482.
PUB. COUNTRY: Sweden
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198305
ENTRY DATE: Entered STN: 19900318
Last Updated on STN: 19900318
Entered Medline: 19830527

AB The **hemodynamics** were monitored during 24 hours in piglets anesthetized with Pentothal-**N2O**/O2, submitted to 33% full-thickness skin burn and resuscitated with 2.4 ml/kg/% burn of 100 mmol NaCl in 2.5% glucose. Three groups were studied: (I) standardized burn, (II) standardized burn and excision after 5 hours, (III) standardized burn, excision and lysine-vasopressin (LVP) given as intravenous infusion in a vasopressor dose. All groups showed similar decrease of cardiac output (CO), which was about 30% 4 hours after burn.

In the two groups with burn excision, however, CO recovery was earlier than in the conservatively treated group I. The improvement was significant between group III and group I. LVP led to higher CO fraction and greater **blood flow** to hepatic artery, reduced flow to proximal gastrointestinal tract and skin and unchanged flow to heart, **kidneys** and other **organs** 24 hours after burn. The mean **blood** loss during and after burn excision was greatly reduced in group III (50 g/25 kg) compared with group II (146 g/25 kg). The therapeutic implications of LVP in excisional burn treatment are discussed.

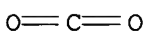
L2 ANSWER 52 OF 52 REGISTRY COPYRIGHT 2006 ACS on STN
RN 109-95-5 REGISTRY
ED Entered STN: 16 Nov 1984
CN Nitrous acid, ethyl ester (8CI, 9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN **Ethyl nitrite (6CI, 7CI)**
OTHER NAMES:
CN N2O
CN Nitrosyl ethoxide
CN Nitrous ether
CN Nitrous ethyl ether
CN **Spirit of ethyl nitrite**
CN Sweet spirit of niter
FS 3D CONCORD
DR 8013-58-9
MF C2 H5 N O2
CI COM
LC STN Files: ADISNEWS, AGRICOLA, BEILSTEIN*, BIOSIS, CA, CAOLD, CAPLUS, CASREACT, CHEMCATS, CHEMINFORMRX, CHEMLIST, CHEMSAFE, CIN, CSCHEM, CSNB, DETHERM*, DIOGENES, EMBASE, GMELIN*, HSDB*, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MRCK*, MSDS-OHS, NIOSHTIC, PDLCOM*, PIRA, PROMT, RTECS*, TOXCENTER, USAN, USPAT2, USPATFULL
(*File contains numerically searchable property data)
Other Sources: DSL**, EINECS**, TSCA**
(**Enter CHEMLIST File for up-to-date regulatory information)

PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

557 REFERENCES IN FILE CA (1907 TO DATE)
2 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
557 REFERENCES IN FILE CAPLUS (1907 TO DATE)
39 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

=>

L6 ANSWER 1733 OF 1734 REGISTRY COPYRIGHT 2006 ACS on STN
RN 124-38-9 REGISTRY
ED Entered STN: 16 Nov 1984
CN **Carbon dioxide (8CI, 9CI)** (CA INDEX NAME)
OTHER NAMES:
CN Carbon oxide (CO2)
CN Carbon-12 dioxide
CN Carbon-12C dioxide-16O2
CN Carbonic acid anhydride
CN Carbonic acid gas
CN Carbonic anhydride
CN Dry ice
CN Khladon 744
CN R 744
FS 3D CONCORD
DR 18923-20-1
MF C O2
CI COM
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, BIOSIS, BIOTECHNO, CA, CABA, CAOLD, CAPLUS, CASREACT, CBNB, CHEMCATS, CHEMINFORMRX, CHEMLIST, CHEMSAFE, CIN, CSCHEM, CSNB, DDFU, DETHERM*, DIOGENES, DIPPR*, DRUGU, EMBASE, ENCOMPLIT, ENCOMPLIT2, ENCOMPPAT, ENCOMPPAT2, GMELIN*, HSDB*, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MRCK*, MSDS-OHS, NIOSHTIC, PDLCOM*, PIRA, PROMT, PS, RTECS*, SPECINFO, TOXCENTER, TULSA, ULIDAT, USAN, USPAT2, USPATFULL, VETU, VTB
(*File contains numerically searchable property data)
Other Sources: DSL**, EINECS**, TSCA**
(**Enter CHEMLIST File for up-to-date regulatory information)



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

197437 REFERENCES IN FILE CA (1907 TO DATE)
806 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
197728 REFERENCES IN FILE CAPLUS (1907 TO DATE)
21 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

17 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L6 ANSWER 1733 OF 1734 REGISTRY COPYRIGHT 2006 ACS on STN
RN 124-38-9 REGISTRY
ED Entered STN: 16 Nov 1984
CN **Carbon dioxide (8CI, 9CI)** (CA INDEX NAME)
OTHER NAMES:
CN Carbon oxide (CO2)
CN Carbon-12 dioxide
CN Carbon-12C dioxide-16O2
CN Carbonic acid anhydride
CN Carbonic acid gas
CN Carbonic anhydride
CN Dry ice
CN Khladon 744
CN R 744
FS 3D CONCORD
DR 18923-20-1
MF C O2
CI COM
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, BIOSIS, BIOTECHNO, CA, CABA, CAOLD, CAPLUS, CASREACT, CBNB, CHEMCATS, CHEMINFORMRX, CHEMLIST, CHEMSAFE, CIN, CSCHEM, CSNB, DDFU, DETHERM*, DIOGENES, DIPPR*, DRUGU, EMBASE, ENCOMPLIT, ENCOMPLIT2, ENCOMPPAT, ENCOMPPAT2, GMELIN*, HSDB*, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MRCK*, MSDS-OHS, NIOSHTIC, PDLCOM*, PIRA, PROMT, PS, RTECS*, SPECINFO, TOXCENTER, TULSA, ULIDAT, USAN, USPAT2, USPATFULL, VETU, VTB
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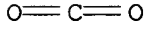
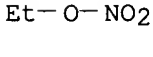
Other Sources: DSL**, EINECS**, TSCA**
(**Enter CHEMLIST File for up-to-date regulatory information)



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

197437 REFERENCES IN FILE CA (1907 TO DATE)
806 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
197728 REFERENCES IN FILE CAPLUS (1907 TO DATE)
21 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L1 ANSWER 473 OF 474 REGISTRY COPYRIGHT 2006 ACS on STN
RN 625-58-1 REGISTRY
ED Entered STN: 16 Nov 1984
CN Nitric acid, ethyl ester (8CI, 9CI) (CA INDEX NAME)
OTHER NAMES:
CN **Ethyl nitrate**
CN NSC 8826
FS 3D CONCORD
MF C2 H5 N O3
CI COM
LC STN Files: ANABSTR, BEILSTEIN*, BIOSIS, CA, CAOLD, CAPLUS, CASREACT, CHEMINFORMRX, CHEMLIST, CHEMSAFE, CSNB, DETHERM*, GMELIN*, HSDB*, IFICDB, IFIPAT, IFIUDB, MEDLINE, MSDS-OHS, NIOSHTIC, PIRA, RTECS*, SPECINFO, TOXCENTER, USPATFULL
(*File contains numerically searchable property data)
Other Sources: EINECS**, NDSL**, TSCA**
(**Enter CHEMLIST File for up-to-date regulatory information)



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

550 REFERENCES IN FILE CA (1907 TO DATE)
2 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
551 REFERENCES IN FILE CAPLUS (1907 TO DATE)
7 REFERENCES IN FILE CAOLD (PRIOR TO 1967)